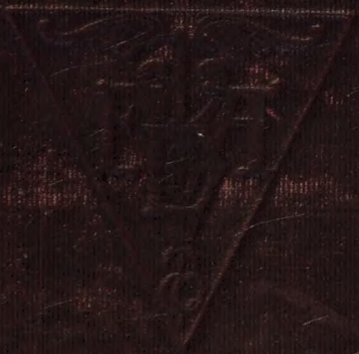

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MODERN
DIAGNOSIS AND TREATMENT
OF
DISEASES OF CHILDREN

A TREATISE ON THE MEDICAL AND SURGICAL DISEASES
OF INFANCY AND CHILDHOOD, WITH ESPECIAL
EMPHASIS UPON CLINICAL DIAGNOSIS
AND MODERN THERAPEUTICS.

FOR

PRACTITIONERS AND STUDENTS OF MEDICINE

BY
HERMAN B. SHEFFIELD, M.D.
(Co-Author of "Practical Pediatrics")
Instructor in Diseases of Children at the New York Post-Graduate Medical School and
Hospital; Visiting Physician (Diseases of Children) to the Yorkville Dispensary
and Hospital for Women and Children, and to the German Poliklinik;
Fellow of the New York Academy of Medicine; Associate
Editor of the Centralblatt f. Kinderheilkunde, etc.

With One Hundred and Fifty Original Half-tone Photo-engravings and Numerous Smaller Illustrations, Some in Colors

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TO

AUGUSTUS CAILLÉ, M.D.
Professor of Pediatrics

AND

HENRY T. BROOKS, M.D.
Professor of Pathology

THIS VOLUME IS AFFECTIONATELY INSCRIBED

As a Tribute to Their Noble Character and High
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PREFACE.

THIS volume is pre-eminently a clinical treatise on the medical and surgical diseases of infancy and childhood, embodying also the essentials of the theory of pediatrics so as to meet the needs of both the medical student and general practitioner. It is based chiefly upon the author's very extensive personal experience in hospital, dispensary and private practice, and presents an amplitude of observations, which is obtainable only in large cosmopolitan centers of population like Greater New York.

The time-worn, stereotyped, verbatim quotations of different authors, and the customary overabundance of illustrations of fads and fancies of enterprising tradesmen—which so conveniently help to "pad" a great many of our text-books—have been entirely eliminated. The valuable space thus gained has been utilized for an ample array of carefully selected photographs and notes to illustrate and emphasize the most important diseased conditions as met in actual practice.

In addition to less important innovations throughout the book, the author has ventured to introduce material changes, especially in the following subjects: Feeble vitality of the newly born; hæmorrhœa congenita et acquisita; microcephalus; infant feeding; gastroenterocolitis; acute meningitis; tuberculosis and tetanism. Furthermore, special chapters are devoted to physical diagnosis and semeiology, general and special therapeutics, congenital malformations, and mental diseases—matters of great importance which have thus far received but little attention in current pediatric literature.

Finally, side-notes are appended to enable especially the medical student at a glance to grasp the most essential points of interest of the subjects under discussion.

We are greatly indebted to our publishers, F. A. Davis Company, for their unselfishness and unusual skill with which they have executed the numerous suggestions of the author in the general make-up of the book.

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CHAPTER I.

Examination of the Patient.

SYSTEMATIC and thorough examination of the patient is the keynote to successful diagnosis and treatment. In infants particularly the physical examination calls for a great deal of patience, care, and scrutiny, and while gentleness in handling the patient is certainly to be preferred, ofttimes firmness will succeed, where kindness utterly fails.) Before, or while, proceeding with the physical examination of the patient, an effort should be made to be informed on the following points of interest:—

fact ?
[illegible]

Family history—

Longevity of the parents, brothers and sisters; the diseases they suffered from, especially as to tuberculosis, rheumatism, heart, kidney or liver disease, alcoholism, epilepsy, insanity, etc. Miscarriages in the mother.

Past personal history of the patient—

Degree of maturity at birth, and mode of delivery (instrumental or otherwise); condition soon after birth, particularly as to signs of traumatism, convulsions, asphyxia, deformity, hemorrhages, skin eruptions, nasal catarrh ("snuffles"); the diseases the patient suffered from at a later period, *e.g.*, gastro-intestinal, exanthematous, pulmonary; otitis, rheumatism, bone affections, etc. Mode of feeding (breast or bottle); gain or loss of weight;[1] time of eruption of temporary or permanent teeth; the time when the patient began to sit up, stand, creep, and walk. Peculiarities of temper, etc.

---

[1] See page 57.

Present history of the patient—

Age of patient.
Mode of onset of the disease (gradual or sudden).
Fever (continuous, remittent or irregular).
Convulsions[2] (apparent cause; time of occurrence; duration).
Vomiting[3] (during, after, or between meals; appearance of vomit).
Skin eruption (location, duration; desquamation).
Diarrhea[4] (duration; frequency and appearance of the stools).
Constipation[5] (acute or habitual; appearance of the stools).
Pain (situation, duration; degree of severity).
Cough[6] (duration; paroxysmal or croupy; appearance of sputum).
Dyspnea[7] (worse after fatigue or at night; sudden).
Cyanosis[8] (duration; mode of onset,—with convulsions).
Urinary disturbance[9] (enuresis, dysuria; suppression; appearance of urine).
Disturbance of sleep (pavor; snoring; twitching).
Behavior and mental capacity (recent change; truancy).
Condition of special senses[10] (defective vision, hearing, etc.).

Intelligent response to the aforementioned questions on the part of those in charge of the patient, will materially aid in the diagnosis.

It will be found of advantage to keep a brief but comprehensive record of the history and condition of the patient at the time of examination, and of the further course of the disease. The different so-called card systems in vogue generally answer this purpose admirably, especially in private practice.

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[2] See page 52.
[3] See page 41.
[4] See page 42.
[5] See page 42.
[6] See page 27.
[7] See page 24.
[8] See page 7.
[9] See page 44.
[10] See pages 8, 9 and 10.

PHYSICAL EXAMINATION.

The history-taking completed, we next turn to the physical examination of the patient. This should be systematic, preferably with the child entirely undressed, and if deemed necessary, should include inspection, palpation, auscultation, percussion, mensuration and weighing.

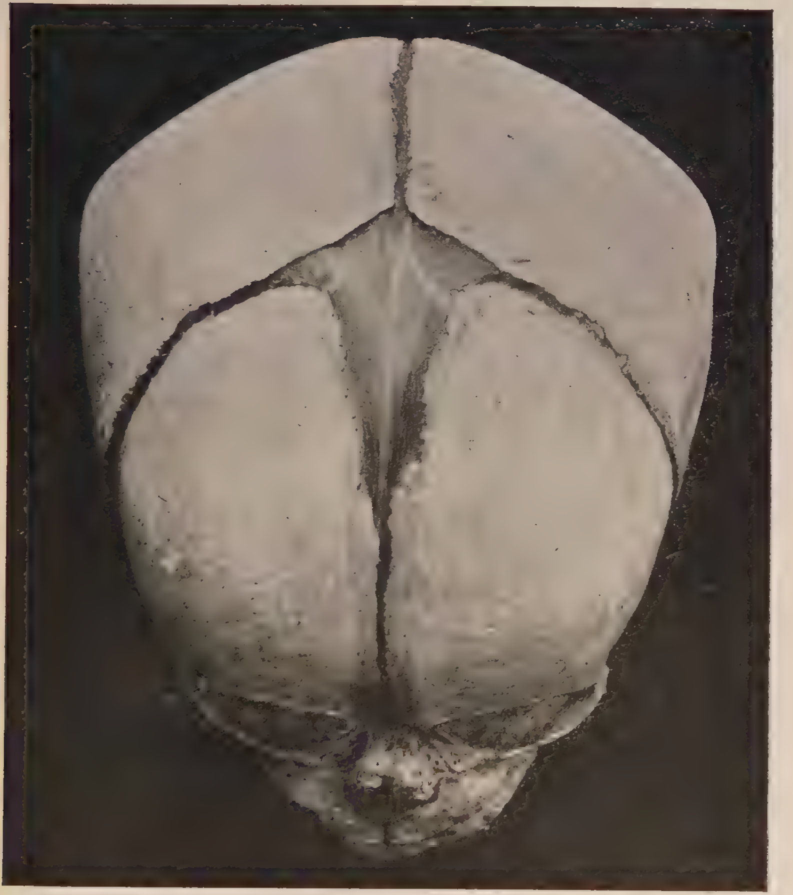


Fig. 1.—Top View of the Fœtal Skull (showing the Anterior Fontanelle and the Frontal, Coronal, and Sagittal Sutures). (*Grandin, Jarman* and *Marx.*)

We usually begin with the examination of the head, noting its size and shape, the condition of the bones of the skull, its fontanelles and sutures, its attitude; facial expression and hue; the condition of the nose, eyes, ears, mouth, lips, tongue, teeth and pharynx.

THE HEAD.

The head is rarely normal in *shape* immediately after birth. The scalp is swollen, the bones are often displaced, and here and there are bruises and ecchymoses, the results of a long and painful journey. Within about a week, the swelling subsides, the bones adjust themselves, the head becomes round or oval and smooth except for the markings of the fontanelles and sutures.

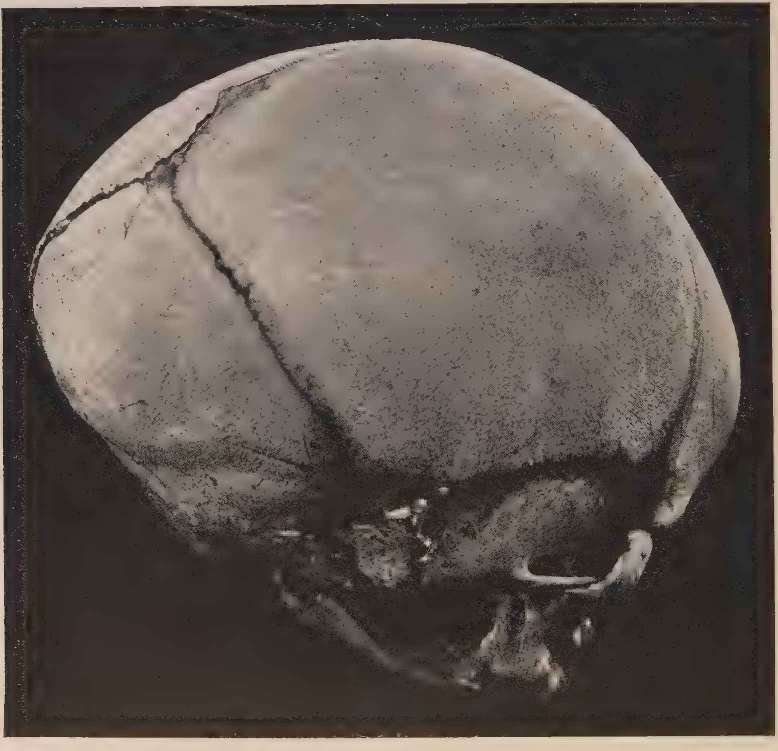


Fig. 2.—Posterior View of the Fœtal Skull (showing the Posterior Fontanelle and the Lambdoidal and Sagittal Sutures). (*Grandin, Jarman* and *Marx.*)

The *cranial circumference* (fronto-occipito diameter) soon after birth measures about thirteen inches. The skull enlarges rapidly up to six months old—seventeen inches; then more slowly about one inch every year up to five years—twenty-one inches; it then remains stationary in growth up to adult life, when it measures from twenty-two to twenty-three inches.

The posterior *fontanelle* closes by the end of the second month, the anterior when the infant is about eighteen months old, at the latest.

A healthy baby is able to hold the head erect when about four months old.

The **skull** is—

Asymmetrical, with depressions and protrusions, in caput succedaneum; meningo- and encephalo-cele; syphilis; neoplasms; abscesses, etc. Hernia of brain.

Large, in hydrocephalus; hypertrophy of the brain; rachitis.

Small, in microcephalus; porencephalia. [illegible]

The **fontanelles** are—

Closed late, in hydrocephalus; rachitis; cretinism; idiocy; osteogenesis imperfecta.

Closed prematurely, in microcephalus; atrophy of the brain.

Distended, in active and passive congestions of the brain, *e.g.*, diverse forms of meningitis; meningismus; hydrocephaloid; intracranial tumors; cerebral hyperemia.

Sunken, in wasting diseases; after great loss of body fluids; after lumbar puncture.

The **cranial bones** are—

Soft and thin, in chronic hydrocephalus; craniotabes.

Hard and thick, in syphilis; exostosis.

The **sutures** are—

Widely separated, in hydrocephalus; intracranial tumors.

Prematurely closed, in microcephalus.

Attitude. The Head is—

Retracted, in general debility; macrocephalus; hydrocephalus; amaurotic family idiocy.

Spasmodically retracted (opisthotonos), in meningitis; meningismus; encephalitis; apical pneumonia.

Turned laterally, in torticollis; hematoma of the sternocleidomastoid muscle; retropharyngeal abscess; cervical spondylitis; cervical adenitis; mastoiditis.

Moving irregularly, in hyperpyrexia; spasmus nutans; chorea; habit spasm.

THE FACE.

1 **Facies dolorosa**—

Continuous pain (eyes open, face wrinkled, mouth half closed and drawn to one side, moaning and whining), in diverse acute inflammatory diseases, *e.g.*, pneumonia, pleurisy, rheumatism, appendicitis.

Intermittent pain (face distorted, red, perspiring; loud crying, tossing, kicking), in colic, dysuria, etc.; vertebral caries ("starting pain").

Different [illegible]
urination.

2 **Facies luctuosa**—

Face of sorrow (forehead and face wrinkled, face pale, emaciated, indifferent, apathetic, eyes half closed) in chronic wasting diseases, especially tuberculosis, and last stage of heart disease.

Facies anxiosa—

Face of anxiety (eyes glistening, congested, red or livid, and perspiring; alæ nasi active) in orthopnea from various causes, *e.g.*, laryngeal stenosis, extensive pneumonia, pulmonary edema; in hysteria.

difference [illegible] when [illegible]

Facies Hippocratica—

Face of grave abdominal distress, or extreme exhaustion (face pale, contracted, corneæ dull, eyeballs and temples deeply sunken, nose pinched, lips dry, cyanotic, and covered with sordes), in moribund state, collapse, cholera nostras, peritonitis, etc.

Facies meningitidis—

Face of internal convulsions (staring look into distance, glassy corneæ, rapidly changing complexion of the face), in meningitis; severe eclampsia.

Facies senilis—

Face of extreme old age (shriveled facial muscles and skin, pointed nose, lusterless eyes), in marasmus; syphilis; chronic hydrocephalus.

4. **Facies idiotes**—

Face of the mentally defective (senile features, open mouth, protruding tongue) in all forms of idiocy and imbecility; less marked in adenoids.

Facies sardonica—

Face of facial muscular spasm (peculiar "grin," proboscisform mouth, sometimes foamy) in tetanus and similar prolonged convulsive conditions.

See also "Facial Paralysis," "Facial Hemiatrophy," "Pertussis."

9 **Facial hue**—

Livid, in congenital and acquired heart disease; in pronounced respiratory difficulty, *e.g.*, laryngeal stenosis, pulmonary edema, asthma, etc.; in cerebral hyperemia; sinus thrombosis; in "holding the breath."

Pale, in anemia; in acute and chronic wasting diseases; sudden pallor, in collapse, *e.g.*, from exhausting hemorrhage.

Waxy, in chronic malaria; suppurative processes; chronic nephritis; malignant disease.

Yellow, in icterus neonatorum or catarrhalis; congenital obliteration of the bile duct; in Buhl's or Winckel's disease; in liver affections, especially due to syphilis.

Purplish, in phthisis pulmonalis ("hectic flush"), hyperpyrexia; pneumonia; compensating heart disease.

Greenish, in chlorosis.

Copper-color (*e.g.*, on forehead), in syphilis.

Bronze color, in Addison's disease.

See also "Exanthemata" and "Skin Diseases."

THE EYES.

The **eyelids** are—

Edematous, without local inflammation, in anemias; heart and kidney diseases; pertussis.

Crusty, red and swollen, in acute and chronic inflammation of the eyelids; in pediculosis of the eyelashes; in congenital syphilis (in conjunction with rhagades at the canthi, and purulent nasal discharge); in scrofulosis (with keratitis, excoriation of the upper lip, and adenitis); red and watery, in nasal catarrh, hay fever, and measles.

Retracted, inability to lower upper lid, from loss of power in the palpebral muscles, in facial paralysis.

Drooping (ptosis) of upper lid, from inability to raise it, in congenital defects of the palpebral levators or their nerve supply; in local trauma; in oculomotor paralysis.

Spasmodically contracting, in local inflammatory processes of the lids; in spasmodic affections, such as chorea and tic; in divers forms of meningeal irritation.

The **eyeballs** are—

Congested, in inflammatory processes of the eye, *e.g.*, keratitis; in meningitis; asphyxia.

Protruding, in exophthalmic goiter; in neoplasms (gumma); in chloroma (frog-like appearance).

of gumma - gum- "cancer" - usually of head -

Immobile, partially or completely, in ophthalmoplegia.

Turned laterally (strabismus, squint); in errors of refraction; in paralysis of the abducens (convergent strabismus); in paralysis of the oculomotor (divergent strabismus—with ptosis, mydriasis, and diplopia).

double vision

Oscillating (nystagmus), in hereditary ataxia; lesions of the corpora quadrigemina; multiple sclerosis; meningitis; sinus thrombosis; hydrocephalus.

sinuses dura mater [illegible] - [illegible]

The **pupils** are—

Contracted, unilaterally, in paralysis of cervical sympathetic, *e.g.*, migraine, cervical rib; in pressure by central tumor. Bilaterally, in affections of the cervical cord, both sides; early stage of meningitis; from the effects of opium and its derivatives, chloral, pilocarpin, physostigmin, etc.

Dilated, unilaterally, in irritation of the cervical sympathetic, *e.g.*, migraine; in oculomotor paralysis. Bilaterally, in marked dyspnea; collapse; from the effects of atropine, belladonna, hyoscyamus, cocaine, etc.

Unequal, in unilateral contraction or dilatation, as aforementioned; in unilateral pontine lesion, and in apoplexy.

Immobile, in adhesions of the iris to the lens; in eclampsia; in lesions of the corpora quadrigemina; in tabes dorsalis (immobility to light, but responding to accommodation—Argyll-Robertson pupil).

Vision is— contraction, esp'l of pupil, dilat[illegible] of pupil

Diminished, in errors of refraction; miosis; mydriasis; hysteria; acute eye affections, *e.g.*, iritis, retinitis, etc.; in corneal opacities, etc.; congenital eye defects, *e.g.*, albinism, irideremia; in toxic amblyopia, *e.g.*, overdoses of quinine, tobacco; congenital amblyopia (usually unilateral); optic neuritis.

Lost, temporarily or permanently, in uremic, diabetic, or other forms of toxemia; in severe convulsions of central origin; congenital cataract; amaurotic family idiocy (gradual onset); in embolism of the central retinal artery (unilateral); local injuries; optic atrophy.

Double (diplopia), in peripheral palsies of the eye muscles, *e.g.*, after diphtheria, influenza, herpes zoster ophthalmicus (unilateral); in strabismus. In orbital palsies, through outside pressure, *e.g.*, neoplasms. In central palsies (affecting the eye on the opposite side). In nuclear palsies, *e.g.*, of the abducens (involving the eye on the same side).

Half, *i.e.*, blindness of one-half of the visual field (hemianopia): lateral or homonymous, in lesions of the optic tract between chiasm and cortex; temporal, in disease of the optic chiasm affecting the anterior or posterior angles; nasal, in disease of the chiasm affecting the outer angles. (See Fig. 3.)

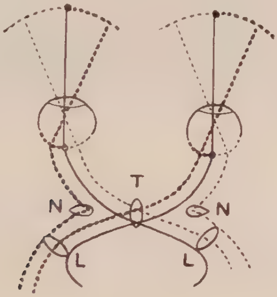


Fig. 3.—Diagram of the Visual Tract. *N.* Lesions producing nasal hemianopia. *L.* Lesions producing lateral hemianopia. *T.* Lesions producing temporal hemianopia. (*Sheffield.*)

THE EARS.

Abnormalities of the ear and adjacent structures—

Asymmetry of the ears, in congenitally, mentally defectives.

Tumefactions, at and about the ear: In the external meatus, in furuncles, abscesses, and local traumatism. In front of the ear, in epidemic parotiditis (often bilateral, though not simultaneously); in secondary parotitis (complicating diseases of the mouth; local infection in the vicinity; acute infectious diseases, *e.g.*, typhoid); in new growths. Behind and downward, pushing the auricle forward, in mastoiditis; in perforating abscess of the external auditory canal; in preauricular lymphadenitis; and much less marked in glandular fever.

Hearing is—

Diminished, at a distance, but not by bone conduction, in external and middle ear disease; in occlusion of the auditory canal by foreign bodies, *e.g.*, cerumen, furuncles; or outside tumors, *e.g.*, parotitis; in nasopharyngeal disease, *e.g.*, adenoids.

Lost, temporarily or permanently, both at a distance and by bone conduction, in congenital defects of auditory apparatus; in compression (by intracranial tumors) or atrophy of the auditory nerve; in disease of the pons or cerebellum which has spread to the fourth ventricle; in amaurotic family idiocy (late).

Disturbed by noises (tinnitus aurium), in foreign bodies in the auditory canal, *e.g.*, cerumen, mycosis, myringitis; in catarrh of the Eustachian tube; in otitis media; neuroses; epilepsy, and mental affections.

THE NOSE.

Abnormalities of the **nose** in structure and function—

Saddle-shaped, sunken, in hereditary syphilis; in traumatism.

Compressed and pointed, in nasal obstruction, chiefly adenoids.

Pinched and pale, in collapse; sudden fright; phthisis pulmonum.

Purplish in color, in circulatory and respiratory difficulties, *e.g.*, pneumonia, heart disease.

Hyperactivity of the alæ nasi, in grave dyspnea.

Nasal voice or cry, in nasal obstruction, *e.g.*, in adenoids, rhinitis, retropharyngeal abscess; in diphtheritic paralysis; in ulceration of the nasal bones, especially in syphilis.

Nasal discharge—

Serous, transparent, later mucous, in acute simple rhinitis ("cold"); measles; hay fever.

Serosanguinolent, later purulent, in diphtheritic, scarlatinal, and syphilitic rhinitis; in the presence of foreign bodies in the nose; in scrofulosis.

Mucopurulent or purulent, in severe acute rhinitis; in putrid infection.

Hemorrhagic (epistaxis), in nasal trauma; inflammation of the nasal mucosa; nasal polypus; adenoids; hemophilia; vicarious menstruation; passive congestion of the brain; increased vascular tension, *e.g.,* hyperpyrexia (especially if sudden, as it is apt to be at the onset of exanthematous diseases), heart and lung diseases, pertussis; in diseases of the blood, *e.g.,* sepsis; leukemia, etc.

THE LIPS.

The lips are—

Excoriated (upper lip) from acrid nasal discharge, in acute and chronic affections of the nose, *e.g.,* rhinitis, adenoids; in scrofulosis; syphilis.

Covered by herpes, a vesicular eruption (usually upper lip at angle of mouth), in ordinary "colds"; in pneumonia; in meningitis cerebrospinalis.

Cracked and scarified, especially at the angles of the mouth, in syphilis hereditaria; but also in burns (usually unilaterally).

Covered by sordes, in septic infections; in typhoid fever.

Rosy in color, in good health.

Deep red, in compensating heart disease.

Purple, in marked dyspnea, from respiratory and circulatory disturbance.

Pale, in divers forms of anemia.

Livid, in heart failure.

Dirty, soot-like, in sepsis; typhoid fever; ulcerative stomatitis.

THE MOUTH.

The mouth is—

Drawn to one side, droops, in facial paralysis, especially when the facial muscles are brought in action; in progressive facial hemiatrophy; in hemiplegia.

Drawn outward and downward, with the lips pointed forward, proboscis-like, in trismus neonatorum, tetanus and tetany.

Broad, grinning, in cretinism; idiocy.

Large from birth, in macrostoma. [illegible]

Small and contracted, in microstoma; in congenital syphilis; from the effects of burns. abnormal smallness

Open habitually ("mouth-breathing"), in nasal obstruction; adenoids; idiocy; in retropharyngeal abscess.

Twitching spasmodically, chorea; habit spasm.

Fœtor ex ore—

Stale insipid, in catarrh of the nasopharynx; dental caries; in febrile diseases; chronic dyspepsia.

Putrefactive, at short range, in diverse forms of simple stomatitis; acute indigestion. At a distance, in noma; malignant diphtheria or scarlatinal angina.

Sulphuretted hydrogen odor, in fetid bronchitis; pulmonary gangrene.

Aceton odor, in diabetes; cyclic vomiting.

Ammoniacal odor, in uremia.

Chloroform, ether, alcohol, etc., odors, from the effects of these drugs.

THE ORAL CAVITY.

In irritable children it is preferable to postpone the examination of the mouth-cavity until the other portions of the body have been thoroughly examined, since the undue excitement usually created by the inspection and palpation of the mouth and throat of the patient greatly interferes with erudition of the other physical phenomena. Through daily practice, the physician soon learns almost at a glance to distinguish the abnormal from the normal; until he has acquired this skill, however, he should examine the contents of the oral cavity slowly and systematically.

The gums, teeth, floor and roof of the mouth; the tongue, buccal mucous membrane, the uvula, fauces, tonsils and posterior pharynx—all should receive careful attention.

The **gums** are—

Whitish, thin, and hard, normally in early infancy.

Reddened, slightly swollen and painful to touch, before eruption of teeth. Disease char. by hem. into

Spongy, swollen, and prone to bleed, in divers forms of mouth stomatitis; in scurvy; purpura; in other grave constitutional diseases, such as leukemia.

Purulent, receding from the teeth, in pyorrhea alveolaris (Riga's disease); alveolar abscess.

Bleeding, without inflammatory symptoms, in hemophilia.

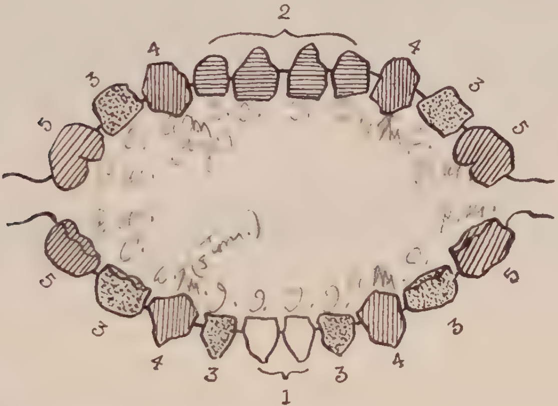
Colored blue, forming a blue line along the margin of the gum, in lead poisoning.

The temporary teeth are twenty in number, and under normal conditions generally appear in groups, at variable intervals, as follows:—

Two lower central incisors at the age of from 6 to 8 months.

Four upper incisors (2 central, 2 lateral), from 8 to 10 months.

Two lower lateral incisors, from 11 to 12 months.



1 ☐ Lower Incisors—fifth to ninth month.
2 ▤ Upper Incisors—eighth to twelfth month.
3 ▒ Lateral Incisors and First Molars—twelfth to eighteenth month.
4 ▥ Stomach and Eye Teeth—eighteenth to twenty-fourth month.
5 ▨ Second Molars—twenty-fourth to thirtieth month.

Fig. 4.—First (Milk) Set of Teeth. (*Starr.*)

Four anterior molars (2 upper, 2 lower), from 14 to 16 months.

Four canines (2 upper, 2 lower), from 18 to 20 months.

Four posterior molars (2 upper, 2 lower), from 22 to 30 months.

Abnormal teething—

Dentitio tarda, *i.e.*, considerable retardation (absence of a tooth at the age of a year or later), in rickets; general debility; congenital syphilis; cretinism; idiocy, etc.

Dentitio precox is of no special significance. Occasionally occurs in congenital syphilis (a tooth may appear soon after birth); in hydrocephalus.

Irregular implantation, the same as in "dentitio tarda" (*q. v.*).

The **permanent teeth** appear normally in the following order:

Four first molars (2 upper, 2 lower) at about 6 years.
Four central incisors (2 upper, 2 lower) at about 7 years.
Four lateral incisors (2 upper, 2 lower) at about 8 years.
Four anterior bicuspids (2 upper, 2 lower) at about 9 years.
Four posterior bicuspids (2 upper, 2 lower) at about 10 years.
Four canines (2 upper, 2 lower) at about 11 years.
Four second molars (2 upper, 2 lower) at about 12 to 15 years.
Four third molars (2 upper, 2 lower) at about 17 to 25 years.

Abnormalities of the **permanent teeth**—

Increased vulnerability and brittleness, in divers grave constitutional affections, *e.g.*, rickets, profound anemia; in neglect and injury of the teeth, especially by escharotic drugs for cleansing of the teeth or medicinal purposes (*e.g.*, the tincture chlorid of iron, acids).

Asymmetry, in hare-lip; cretinism and other forms of defective mentality; nasal obstruction, "mouth-breathing"; thumb sucking.

Looseness, in gingivitis; ulcerative stomatitis; mercurialism; scurvy; pyorrhea alveolaris.

Hutchinson teeth, *i.e.*, peg-shaped, dwarfed upper central incisors, notched in their cutting edge, in inherited syphilis.

The **floor** of the **mouth** may present—

Adhesio linguæ, a frequent cause of difficult suckling; and later of difficult speech.

Sublingual ulcer, in protracted coughing, especially pertussis.

New growths, *e.g.*, ranula, fibroma sublingualæ; salivary calculi; inflammatory swelling.

The **palate** is—

Highly arched and asymmetrical, in divers forms of mental degeneracy; adenoids.

Defective, or perforated, in congenital clefts of the palate;

in syphilitic or gangrenous processes (*e.g.*, diphtheria, scarlatina).

Red, velvety, in scarlatina.

Punctiform or stellate, in measles or rötheln.

Vesicular with red areola, in chicken-pox.

Papular, in small-pox.

Whitish-yellow eroded dots, in Bednar's aphthæ.

Minute, yellowish-white milia, in "epithelial pearls." [illegible] in Som

White specks or scattered patches, in different forms of stomatitis.

Hemorrhagic and punctiform, in hemorrhagic diathesis; tuberculous and cerebrospinal meningitis; pernicious blood affections.

The **buccal mucous membrane** presents, in addition to the discolorations occurring upon the palate, also the following:—

Brownish, greenish or grayish ulcer, in incipient noma.

Red spots with central, rounded, slightly elevated, bluish efflorescence (Koplik-Filatov spots), in measles.

The **tongue** is—

Large, in congenital macroglossia; in cretinism; idiocy; glossitis.

Furred, in all acute and protracted forms of gastro-enteritis; febrile diseases; nasopharyngeal catarrh.

Red, in scarlatina (strawberry tongue); stomatitis; glossitis; gastritis (hyperacidity).

Yellow, in biliousness; liver disease.

Pale, in anemia.

Gray, brown and somewhat black, with red border and tip, in typhoid fever; in sepsis.

Black, in profound sepsis; in collapse impending death.

Livid, in general cyanosis; congenital heart disease.

Spotted, desquamating, in geographical tongue; hyperpyrexia; stomatitis. geogra

Fissured, in glossitis desiccans; hyperpyrexia; burns.

Ulcerated, in severe forms of stomatitis; in syphilis; tuberculosis; traumatism (biting of the tongue during an epileptic fit; irritation by carious teeth).

Dry, in mouth-breathing; excessive thirst (*e.g.*, hyperpyrexia, diabetes); in sepsis.

Protruding, in macroglossia (*e.g.*, idiocy, cretinism).

Drawn to one side, in paralysis of the hypoglossal nerve (towards the diseased side); in peripheral facial palsy (towards the healthy side).

Tremulous, in hyperpyrexia; debility; chorea; disseminated lateral sclerosis; bulbar paralysis.

The **saliva** is—

Increased in quantity, in mercurialism; stomatitis; teething; idiotic conditions, and adenoids.

Diminished in quantity, in fever; from the effects of atropine, etc.; parotitis; glossitis.

The **uvula**—

May be elongated; the seat of a deposit which may extend from the tonsils or from the buccal mucous membrane (*e.g.*, stomatitis).

The **tonsils** are— tonsillitis,

Enlarged, in divers form of amygdalitis; diphtheria; scarlatina; pharyngitis; influenza; rheumatism; abscess; traumatism; glandular fever; foreign bodies (*e.g.*, calculi); new growths (*e.g.*, fibrous polypus, hydatid cyst).

The seat of a deposit, in follicular tonsillitis (small isolated white pellicles which coalesce); in parenchymatous tonsillitis (at first white, later yellowish green, resembling "point of abscess"); in tonsillitis herpetiformis (vesicular deposit, ending into ulcer); in necrotic tonsillitis (yellowish-green patch); in influenza and pharyngitis (superficial exudation); in scarlatina and diphtheria (large pseudomembrane); in stomatitis mycotica (flour-like deposit).

In doubtful cases it is imperative to examine a smear of the tonsillar deposit microscopically or bacteriologically.

THE NECK.

The **lymphatic glands** are—

Enlarged, in all forms of angina, especially that due to diphtheria or scarlet fever; in affections of the mouth (*e.g.*, stomatitis, gingivitis); in parotitis; mastoiditis; glandular fever; pseudoleukemia; scrofulosis (tuberculosis); eczema capitis; local infections.

The **thyroid gland** is—

Enlarged, in goiter; exophthalmic goiter; endemic goitrous cretinism; thyroiditis; temporarily before menstruation.

Atrophied or absent, in sporadic cretinism.

Tumefactions (other than those of the glands of the neck)—

Hematoma of the sternocleidomastoid, in the center or at sternal insertion of the sternomastoid muscle.

Hygroma cysticum, between lower jaw and clavicle, attains enormous size.

Fistula coli congenita, at sternoclavicular articulation.

Pulsation of the—

Arteries, in aortic regurgitation; hyperpyrexia, etc.

Veins, in tricuspid insufficiency.

Stiffness of neck—See "Attitude of Head," page 5.

THE THORAX AND ITS CONTENTS.

AUSCULTATION AND PERCUSSION.

Auscultation is best performed by means of a small bi-aural speculum, as with this instrument every inch of the infantile thorax can be thoroughly examined and small circumscribed lesions readily detected.

Normally the respiratory sound is puerile (rough vesicular) in infancy or early childhood; and vesicular in older children.

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nurse
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In auscultating the infantile *lungs* we should remember the following peculiarities:+1. During quiet respiration the inspiratory sound is fairly audible, while the expiratory sound is but slightly so,—hence to obtain more distinct physical signs it is of advantage to disturb the infant, or to make it cry. + 2. Owing to the larger diameter of the right bronchus, the respiratory sounds are louder on the right side than on the left. —3. Pure bronchial breathing is often normally heard over the interscapular regions, especially to the right of the spinal column. 4. Adventitious sounds originating in the nasopharynx and larynx are frequently transmitted to the chest and may be misinterpreted as signs of pulmonary disease.

The normal pulmonary percussion note is clear, loud, and somewhat tympanitic. It is somewhat metallic, when the child cries; cracked-pot-like, over the right subclavicular region; somewhat dull over the areas overlapping the liver, heart and spleen.

Percussion of the infantile *lungs* should be practised while the patient is held in a sitting posture, perfectly still and as erect as possible. It should be performed gently, preferably during the height of inspiration and expiration. Every portion of the lung should be carefully gone over, paying especial attention to the sub- and supra-clavicular spaces, which are not rarely the seat of consolidation, and the area corresponding to the tracheal bifurcation, which is often the seat of tuberculization of the bronchial glands. The physical signs obtained on percussion are not always conclusive, if percussion is performed too forcibly (may give rise to covibration of the more distant parts); if the child cries (during the act of crying compression of the lung by ascension of the diaphragm produces artificial dulness); if the position of the child is faulty (*e.g.*, lying on the abdomen pushes the diaphragm upward and compresses the lungs); or if the thorax is bent sharply forward.

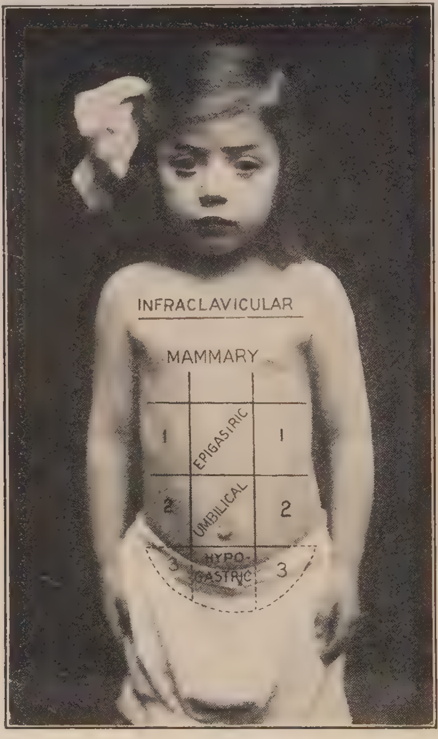

Fig. 5.—The Thoracic and Abdominal Regions. 1. Hypochondriac. 2. Lumbar. 3. Inguinal. (*Sheffield.*)

In auscultating the *heart* we should bear in mind the following: 1. Accentuation of the first sound is heard equally as well at the arterial and venous orifices. 2. Accentuation of the second sound is ordinarily not heard until about the age of puberty. 3. Both heart sounds are louder in children than in adults and are more widely transmitted. 4. Reduplication of the heart sounds is not uncommon, and generally the result of excitement. 5. In infants hemic murmurs are rare. 6. The heart-beat, as to frequency and rhythm, is apt to undergo great variations on slightest provocation.

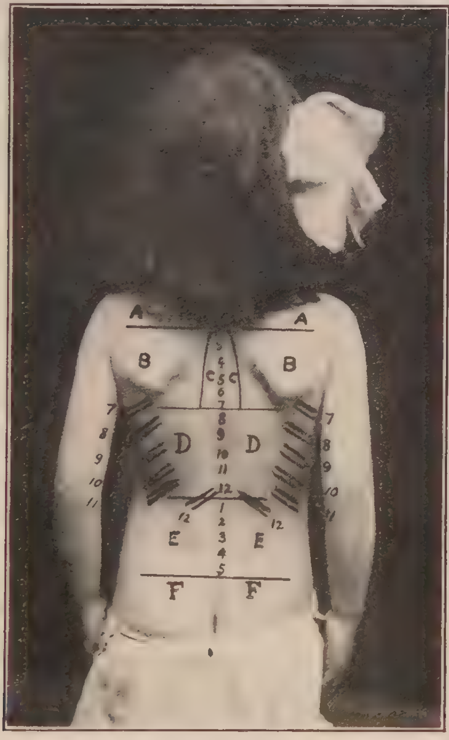

Fig. 6.—The Regions of the Back. *A.* Suprascapular or supraspinatus. *B.* Scapular. *C.* Interscapular. *D.* Infrascapular or lower dorsal. *E.* Lumbar. *F.* Sacral. (*Sheffield.*)

Percussion of the child's *heart* should be performed very gently while the patient sits quietly and bent slightly forward. The data obtained on percussion while the child cries, holds its breath, etc., are not wholly to be depended upon, since during bodily unrest the heart is very apt to change its relation to the chest wall. The same holds true in the event of the heart being overlapped by emphysematous lungs; if the heart is left bare by atrophy of the adjacent lung portions or by retraction of the heart or lungs by pleuritic or pericardial adhesions.

THE THORAX.

The normal infantile thorax is round and somewhat cylindrical, its sagittal and transverse diameters being nearly equal. As the child grows older, the chest assumes a more conical shape, until at puberty it resembles that of the adult. The chest walls of the child are thin, elastic and yielding, owing to incomplete development of the muscular and bony structures. The ribs of the infant are nearly horizontal.

The **measurements** of the thorax are—

In the newly-born infant, about 14 inches.
At one year, 17 inches.
At three years, 20 inches.
At six years, 23 inches.
At twelve years, 26 inches.
At the end of the fifteenth year, the measurement of the circumference of the chest is about half of that of the body length.

Up to about eighteen months the circumference of the chest nearly equals that of the head. If from the end of the second year on the circumference of the head exceeds that of the chest, there is a strong suspicion of hydrocephalus, marked rachitis, contraction of the chest through pulmonary disease or imperfect development (adenoids). On the other hand, if the chest measurement in early childhood greatly exceeds that of the head, it is indicative either of an abnormality of the chest, *e.g.*, distension by fluids, or of congenital maldevelopment of the head, *e.g.*, microcephalus, infantilism.

Abnormal shape of chest—

Barrel-shape (deep, short and broad), in emphysema, and the lung affections that precede it, *e.g.*, asthma, pertussis; protracted laryngeal stenosis.

Flask-shape (flat, narrow and long), in phthisis pulmonum; nasopharyngeal stenosis, especially adenoids.

Funnel-shape (depression of lower portion of sternum), in rachitis; Barlow's disease; also congenital.

Pigeon-breast-shape (protrusion of median portion of sternum), in rachitis; congenital heart disease.

Unilateral bulging, in pneumothorax; pleurisy or pericarditis with effusion; tumor; scoliosis (opposite side).

Unilateral flattening, in pleuritis retrahens (after absorption of fluid); pulmonary contraction, *e.g.*, tuberculosis; after pyothorax operation; scoliosis.

Tumefactions—

Costal, nodular, in rachitis (rachitic rosary); tuberculous and syphilitic processes; multiple exostoses.

Intercostal, doughy, in suppuration of the bronchial glands; empyema necessitatis; lung hernia.

Mammary, in mastitis; cold abscess; as a partial manifestation of parotitis; new growths.

Abnormal shape of scapulæ—

Prominent, uni- or bi-laterally, "angel wing" deformity, in congenital malformation; in emaciation. Unilaterally, in scoliosis; paralysis of the scapular muscles, *e.g.*, after local trauma; poliomyelitis; progressive atrophy.

Sunken, in scoliosis; after empyema operation.

Activity of the thorax in breathing is—

Increased, bilaterally, in asthma; laryngeal obstruction. Unilaterally, on the sound side, in pleurisy with effusion; pneumothorax; fixed deformities.

Diminished, bilaterally, in emphysema; hydrothorax; diffuse tuberculization; paralytic conditions of the chest wall; sclerema; collapse. Unilaterally, in pleurisy with effusion; pneumothorax; pleurodynia; pleuropneumonia with "stitch" pain.

Pain on pressure—

Superficial, in rheumatism of the chest muscles; intercostal neuralgia; affections of the ribs (caries, periostitis, fracture, etc.); localized abscesses (empyema necessitatis); and tumefactions (*e.g.*, mastitis).

Deep, in pleurisy; pneumonia; phthisis pulmonalis.

THE LUNGS.[1]

The lungs are normally fully distended with air within the first few hours of life. In the premature or delicate infant full lung inflation may not occur until several weeks after birth. The lower lobes particularly may remain in a state of atelectasis.

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[1] See "Auscultation and Percussion," page 17.

The normal *boundaries* of the lungs differ somewhat with the age of the child. On both sides they project with their summits into the supraclavicular fossæ. From here they descend in the following manner:—

The **right lung** lies—

In the sternal line at a point corresponding to the fifth (upper border) rib.

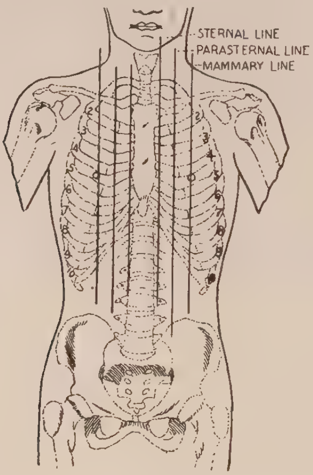

Fig. 7.—Diagnostic Lines of the Thorax. (*Sheffield.*)

In the parasternal line at a point corresponding to the fifth (lower border) rib.

In the mammary line at a point corresponding to the sixth rib.

In the axillary line at a point corresponding to the seventh rib.

In the scapular line at a point corresponding to the tenth rib.

The **left lung** lies—

In the sternal line at a point corresponding to the fourth rib.

In the parasternal line at a point corresponding to the fourth rib.

In the mammary line at a point corresponding to the sixth rib.

In the axillary line at a point corresponding to the seventh or eighth rib.

In the scapular line at a point corresponding to the tenth rib.

Posteriorly the base of the left lung is slightly lower than that of the right lung.

Number of **respirations** per minute—

In the newly born infant from 35 to 40.

At the end of first year, 30.

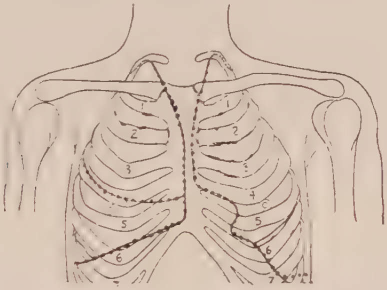

Fig. 8.—Anterior Boundaries of the Lungs. (*Sheffield.*)

At the end of second year, 25.

At six years, 22.

At twelve years, 20. – 17.

Character of **respiration**—

Abdominal, in children under four years of age.

Costo-abdominal, in children (male and female) up to ten years; in the male, in older ones.

Thoracic, in girls over ten years old.

Regularity of respiratory rhythm is usually not fully established before the age of two years.

Abnormalities of **respiration**—

Increased frequency, in respiratory and circulatory diseases (see "dyspnea"); pyrexia; emotional excitement; compression of the lungs by an accumulation of gas, fluids, or solid masses.

Diminished frequency, in grave central disease; extreme weakness; poisoning from belladonna, opium, etc.

Costal breathing in boys over ten years old, and increased costal breathing in girls, in inflammatory diseases of the abdominal and pleural cavities (by interference with the action of the diaphragm) *e.g.*, peritonitis, pleuritis; in abdominal distension by gases, fluids, or solid masses; in paralysis of the diaphragm, *e.g.*, bulbar paralysis, polioencephalitis, neuritis (postdiphtheritic) of the phrenic nerve; in drug poisoning; in hysteria.

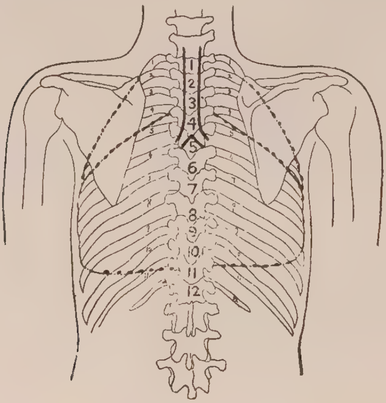

Fig. 9.—Posterior Boundaries of Lungs. (*Sheffield.*)

Purely abdominal breathing, especially in girls over ten years old, in emphysema; scleroderma; paralysis of respiratory muscles, *e.g.*, bulbar paralysis.

Irregular breathing, in conditions associated with "difficult breathing"; in cerebrospinal affections; in atelectasis; painful diseases of the respiratory muscles; in hysteria.

Stertorous breathing, in nasopharyngeal obstruction, *e.g.*, retropharyngeal abscess, adenoids; in uremic or apoplectic coma.

Cheyne-Stokes' breathing, occasionally in infants during sleep; in heart failure from divers causes; in meningitis, especially the tuberculous variety; in meningeal hemorrhage, tumors or abscess exerting pressure upon the brain; in drug poisoning, *e.g.*, opium; in death-agony.

Difficult or labored breathing (dyspnea), in laryngeal, tracheal or bronchial obstruction from divers causes, *e.g.*, croup, diphtheria, large thymus, asthma, etc.; in affections associated with diminution of the usual pulmonary breathing area, such as active or passive congestion, compression or displacement by neoplasms, *e.g.*, pneumonia, pleurisy or pericarditis with effusion, deformities of the thorax, advanced pulmonary tuberculosis; in grave circulatory disturbance inducing deficient oxygenation of the blood or obstruction to pulmonary circulation, *e.g.*, blood, or heart diseases ("cardiac asthma"); in conditions giving rise to "irregular breathing" (*q.v.*) "stertorous breathing" (*q.v.*), and "Cheyne-Stokes' breathing"; in neuroses, *e.g.*, hysteria, neurasthenia—asthma hystericum.

Respiratory sounds—

Vesicular, exaggerated, in bronchial inflammation; atelectasis.

Weak, in thickened pleura; moderate pleuritic effusion; emphysema.

Absent, in extensive pleuritic effusions.

Bronchial, over the seat of the lesion, in pneumonia; tuberculization.

Above the seat of lesion, in compression of the lung by tumors in the chest cavity or by pleuritic exudates.

Amphoric, in smooth-walled cavities; open pneumothorax.

Secretory sounds—

Dry, sibilant and sonorous rhonchi, in bronchitis; asthma (wheezing and whistling).

Dry, crackling, in incipient phthisis (apex); beginning of second stage of pneumonia.

Moist, large and medium-sized râles, in bronchitis (larger bronchial tubes) with abundant secretion; in cavities.

Moist, small râles, in capillary bronchitis.

Moist, crepitant (fine) râles, in croupous pneumonia (crepitatio indux or redux); catarrhal pneumonia; capillary bronchitis (in conjunction with coarse râles); tuberculization; pulmonary edema (in conjunction with larger moist râles).

Metallic tinkling, in pneumothorax.

Metallic splashing or gurgling, in sero- or pyo-pneumothorax.

Friction sound, in pleuritis sicca; pleuropneumonia; miliary tuberculosis. It is not altered by coughing, as is the case with râles.

Vocal resonance[1]—

Diminished, in bronchitis with free secretion; pleurisy with effusion; obstruction of bronchial tube; emphysema; pneumothorax.

Increased, in tuberculization; pneumonia (over-consolidation).

Bronchophony (concentration of voice near the ear), in tuberculization; pneumonic consolidation; compressed lung above pleuritic effusion; bronchial dilatation.

Exaggerated bronchial whisper; the same as for bronchophony (*q.v.*).

Pectoriloquy (complete transmission of sound), the same as for bronchophony (*q.v.*).

Amphoric voice ("the echo"), in large cavity; pneumothorax.

Egophony, bleating (goat-like resonance of voice), in pleurisy with effusion (near upper boundary of dulness); pleuropneumonia; hydrothorax.

Abnormal percussion-resonance—

Dull or diminished resonance, in pneumonia; tubercle; neoplasms; pulmonary gangrene; pulmonary abscess with thick masses; pleuritic thickening; atelectasis.

Flat or absence of resonance, in pleurisy with effusion; hydrothorax; hemothorax. Resonance may alter with change of patient's position. Also in last stage of pneumonia with extensive consolidation.

Tympanitic, or drum-like, resonance, in tuberculosis (cavities); open pneumothorax; lung atrophy; above pericardial or pleuritic exudations or near neoplasms—the result of increased air pressure; pulmonary edema; moderate emphysema.

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[1] Vocal resonance elicited on auscultation corresponds to vocal fremitus as obtained by palpation. Fremitus is increased in consolidation and diminished in effusions.

Amphoric, metallic, or concentrated tympanitic sound, in large tuberculous cavity with solid and tense walls lying close to the chest wall; occasionally heard in healthy child during crying.

Cracked-pot-resonance, in pulmonary cavity communicating with the bronchial tubes—usually in tuberculosis; may be elicited also in healthy child during talking or singing.

Band-box-note (abnormally loud and deep), in pronounced emphysema; pneumothorax with strong tension of the chest wall.

COUGH.

It is essentially a reflex act arising from direct or indirect irritation of the respiratory center. In a measure it can be voluntarily produced or suppressed. The ability to cough is lost in paralysis of the crico-arytenoid or the respiratory muscles, hence cessation of coughing—with plenty of mucus in the bronchial tubes—particularly in pulmonary disease, is considered a bad omen. The nature of the cough may often be decided upon from its character.

The **cough** is usually—

Short and somewhat hoarse, in nasopharyngeal catarrh, adenoids.

Loud and barking, in laryngitis and spasmodic croup.

Dull, barking and somewhat moist, in ulceration of larynx (diphtheria, syphilis, etc.).

Dry, tight and whistling, in early bronchitis.

Soft, deep, and loose, in advanced bronchitis.

Paroxysmal and whooping, in pertussis and other spasmodic affections.

Hemming, in incipient phthisis and in nervousness.

Short, sharp and painful, in pneumonia, pleurisy, and cardiac disease.

Deep and distressing, in chronic phthisis, asthma, emphysema, etc.

Too much reliance should not be placed upon the character of the cough, as it is very apt to vary with the duration of the cough, medication and complications. By far more reliable information may be obtained from a careful examination of the expectoration.

SPUTUM, EXPECTORATION.

In cases where the children cannot or will not expectorate, the sputum may be obtained by introducing into the throat a sterile cotton swab or fenestrated stomach-tube—both of which usually receive enough of sputum during the act of coughing as to suffice for ordinary examination.

The **expectoration** is—

Mucous, frothy, grayish-white, in acute catarrh of the air passages.

Mucopurulent, tenacious, yellowish-gray, in chronic tracheobronchial catarrh; in pertussis (voluminous, often mixed with vomitus); in asthma (Curschmann's spirals, Charcot crystals); in bronchiectasis (periodic "mouthful expectoration," separable into a purulent and mucoserous layer).

Purulent, fetid, dirty grayish-green, in fetid or putrid bronchitis (separable in three layers, suspended in the lowest, purulent, layer are Dittrich's plugs); in pulmonary abscess (separable in two distinct layers, containing a great number of micrococci, elastic fibers, fat-crystals, etc.); in pulmonary gangrene (same as putrid bronchitis, plus tissue-fragments).

Serous, prune-juice-like, and profuse, in pulmonary edema.

Bloody, in nasopharyngeal catarrh with violent paroxysms of coughing (occasional streaks of blood); in foreign bodies in the air-passages (bright red mixed with frothy mucus); in pneumonia (uniformly stained, "rusty" sputum to dark "prune-juice" color with pneumococci); in heart disease, with edema (the same as in pulmonary edema from other causes; besides "heart-cells"); in tuberculous lesions of the air-passages (either large hemorrhage, "hemoptysis," or blood stained "nummular" and heavy sputum, containing tubercle-bacilli); in neoplasms ("red-currant"-like sputum, with characteristic histologic structures); in vicarious menstruation; hemorrhagic diathesis, and hysteria. See "Hematemesis" and "Epistaxis."

The expectoration contains numerous micro-organisms and occasionally bile (in icterus), hydatid hooklets, distomum pulmonale, and cercomonas.

THE HEART.[1]

The heart is comparatively larger in infancy than in later life. It is relatively largest at birth, and smallest at about the age of seven years. At birth the walls of both ventricles are nearly of equal thickness, but as the infant grows older the left ventricle rapidly gains in thickness, so that by the end of the second year it is almost twice as thick as the right ventricle.

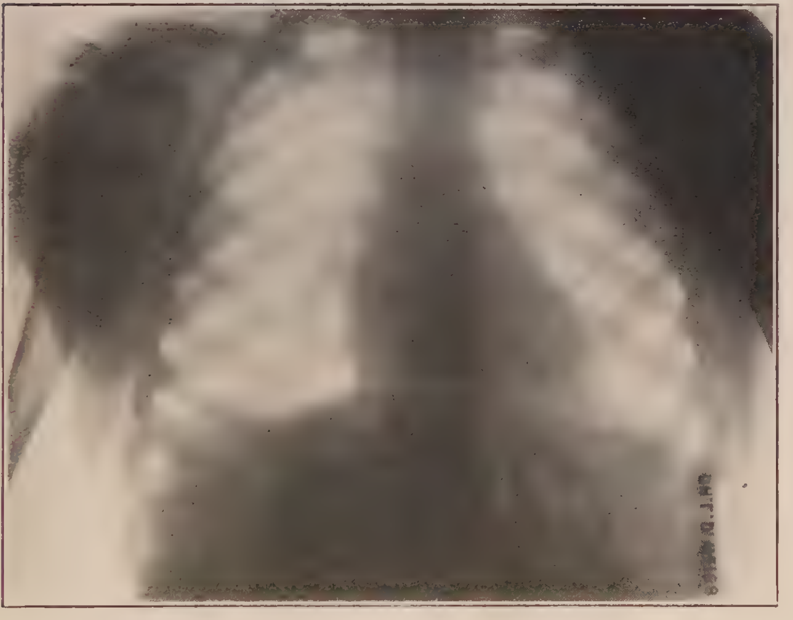

Fig. 10.—Skiagram of Normal Heart of a Child 8 Years Old.

Corresponding to the relatively larger size and more transverse position of the heart of the young child, its boundaries are greatly at variance with those of the heart of the adult.

The **boundaries** of the normal heart—

The apex-beat is situated—

To the left of the mammary line, in the fourth intercostal space up to the fourth year of age.

At the mammary line, at or slightly below the fifth rib up to the eighth year.

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[1] See "Auscultation and Percussion," page 17.

Slightly to the right of the mammary line, in the fifth intercostal space up to the twelfth year.

Between the mammary and parasternal lines, *i.e.,* the same as in the adult, in children over twelve years.

The *relative "heart-dullness"* in infants is bounded as follows:—

Above, by a line corresponding to the lower border of the second rib.

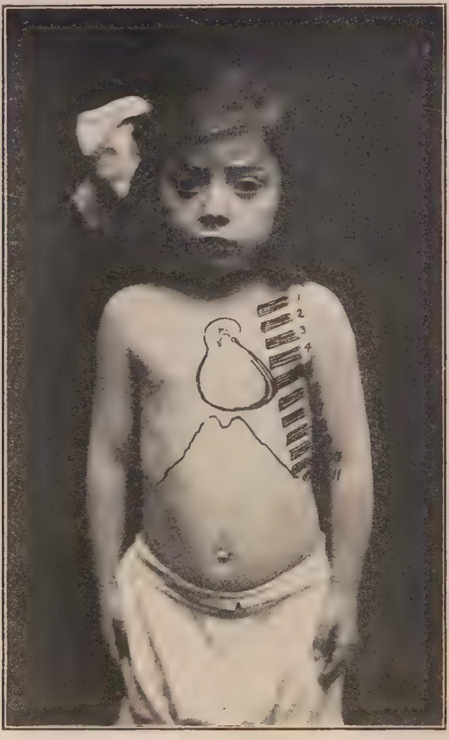

Fig. 11.—Topography of the Heart. (*Sheffield.*)

On the left side, by a line parallel and slightly to the left of the left mammary line.

On the right side, by the right parasternal line.

Below, by a somewhat semicircular line along the fifth rib.

As the child grows older and the heart assumes a more oblique and lower position, the boundaries of the relative heart dullness gradually fall in line with those of the adult.

The *absolute "heart-dullness"* in infants is bounded as follows:—

Above, by the upper border of the fourth rib.

On the left side, by the left mammary line (slightly to the right of it).

On the right side, by the left sternal line.

Below, by a line corresponding to the upper border of the fifth rib.

These boundaries, like those of the relative heart dullness, change gradually with the advance of the child's age, so that in children over twelve years old the upper boundary is formed by the fourth rib, the lower by a line drawn parallel to and between the fifth and sixth ribs, on the right side by the sternal line, and on the left by a line midway between the parasternal and mammary lines.

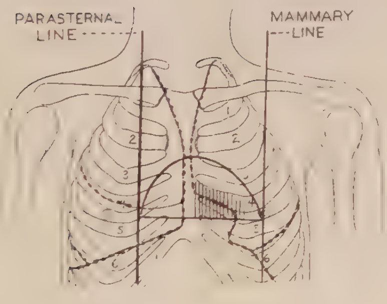

Fig. 12.—Up to 4 years.

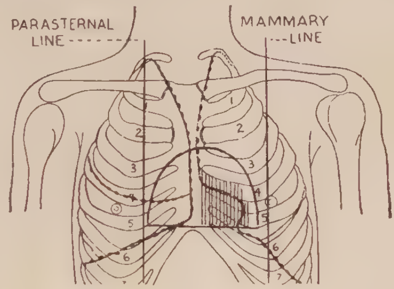

Fig. 13.—Up to 8 years.

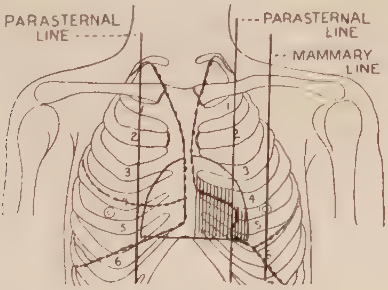

Fig. 14.—Up to 12 years.

The Relative and Absolute Heart Dullness at Different Ages.
(*Sheffield.*)

The normal **pulse-rate** (most reliable when patient is asleep), is—

In the newly-born infant from	120 to 150 per minute.
At one year old	100 to 120 per minute.
At four years	90 to 100 per minute.
At eight years	80 to 90 per minute.
At twelve years	75 to 80 per minute.

Normal **pulse-respiration** ratio is approximately 1:4. A ratio of 1:3 or less is a certain indication of pulmonary disease, especially pneumonia.

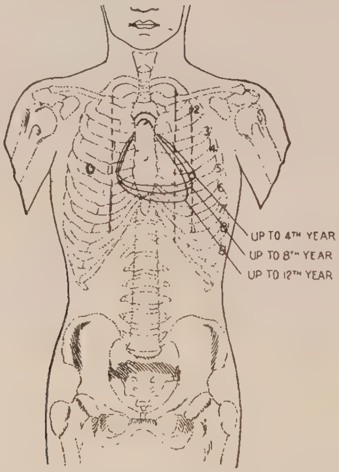

Fig. 15.—Location of Heart-apex at Different Ages. (*Sheffield.*)

Clinit

Apex-beat—

Displaced—

Varies as
[illegible]
of position

Outward, to the left, in hypertrophy of the right ventricle; dilatation of the right ventricle; right-sided pleurisy with effusion; right-sided pneumothorax; abdominal distention, pushing the diaphragm upward and the heart to the left.

Outward and downward, in hypertrophy of the left ventricle; dilatation of the left ventricle; pericardial effusion; congenital or acquired (by pressure from above, *e.g.*, tumor or abscess) dislocation of the heart.

Inward, to the right, in left-sided pleuritic effusion; pronounced left-sided deformity of the thorax; persistence of the embryonic position or situs inversus (up to dextrocardia).

Effaced (*i.e.*, apex-beat is invisible and barely palpable), in obesity; pericardial effusion; heart-failure; emphysema; edema cutis; tumors.

Diffuse, and weak in irregularity of the heart associated with grave heart disease.

Diffuse and strong, in cardiac hypertrophy; hyperpyrexia; overstimulation; excitement. The cardiac impulse may only *appear* strong when the chest wall is very thin.

Heart-sounds—

- Accentuation of—
  - Systolic mitral, in excitement; fatigue; fever; hypertrophy of left ventricle.
  - Diastolic pulmonic, in hypertrophy of right ventricle.
  - Diastolic aortic, in hypertrophy of left ventricle.
- Weakening of—
  - Systolic mitral, in dilatation of the left ventricle; loss of compensation.
  - Diastolic pulmonic, in dilatation of the right ventricle (*e.g.*, relative tricuspid insufficiency); stenosis of pulmonary artery.
  - Diastolic aortic, in aortic stenosis.
- Division (double) of diastolic at apex, in mitral stenosis; adhesive pericarditis.
- Gallop rhythm, in heart-failure (*e.g.*, incipient diphtheritic paralysis).
- Metallic ringing, in pneumopericardium; pneumothorax; large pulmonary cavity; intense meteorism.
- Murmurs—
  - Systolic, loudest at apex and transmitted to axilla and angle of left scapula, in mitral regurgitation.
  - Systolic, loudest at base (midsternum) and transmitted to the arteries upward and sometimes over the whole sternum, in aortic obstruction.
  - Systolic, at base, but *not* transmitted upward, in pulmonic obstruction.
  - Systolic, loudest at ensiform cartilage, in tricuspid regurgitation.

Diastolic, loudest at base, and transmitted to apex and ensiform cartilage, in aortic regurgitation.

Diastolic or presystolic, loudest at apex, in mitral obstruction.

To-and-fro friction, superficial, limited to precordium, not influenced by respiration (as in pleuritis sicca), in fibrinous pericarditis.

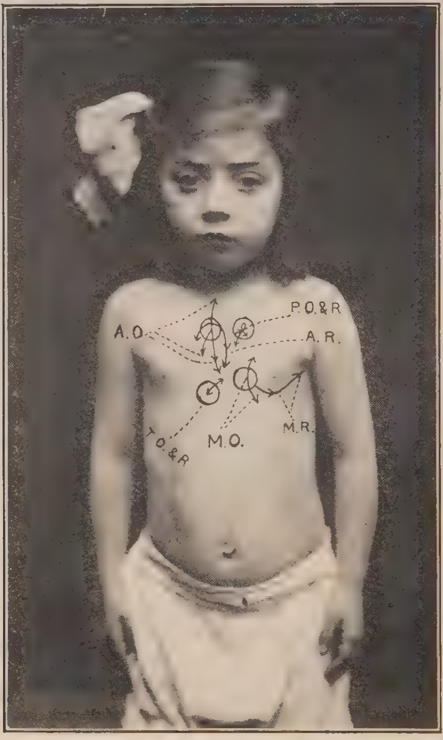

Fig. 16.—Topography of Cardiac Valves. Points of Transmission of Heart-murmurs. *A. O.*, Aortic Obstruction. *P. O.* and R., Pulmonic Obstruction and Regurgitation. *A. R.*, Aortic Regurgitation. *T. O.* and *R.*, Tricuspid Obstruction and Regurgitation. *M. O.*, Mitral Obstruction. *M. R.*, Mitral Regurgitation. (*Sheffield.*)

Omit

Areas of heart-dullness—

Enlarged—

To the left, in hypertrophy or dilatation of the left ventricle.

To the right, in hypertrophy or dilatation of the right ventricle.

Bilaterally, in pericardial effusion. The area of dullness is larger in sitting than in recumbent posture; it is often triangular, wider below than above.

Reduced—

In pulmonary emphysema; pneumopericardium.

Displaced—

In congenital malpositions, *e.g.*, dextrocardia, mesocardia, diaphragmatic hernia.

In acquired affections, such as pneumothorax; pleurisy with effusion; neoplasms; pleuritic retraction; atrophy of the lungs.

The **pulse**— +disease

Frequent, in fright; excitement; fear; febrile diseases (except uncomplicated typhoid or meningitis); valvular heart diseases (except aortic stenosis); anemias, especially on slight exertion; tachycardia; exophthalmic goiter; convalescence from acute affections; paralysis of the heart (central or peripheral paralysis of pneumogastric nerve); heart-failure (*e.g.*, collapse in febrile diseases).

Slow, in uncomplicated typhoid fever or meningitis; after crises (*e.g.*, pneumonia); acute nephritis; catarrhal jaundice; intracranial pressure (*e.g.*, hydrocephalus, hemorrhage, tumors); heart disease, such as aortic stenosis, myocarditis; bradycardia; profuse hemorrhage; marked inanition (*e.g.*, a pyloric stenosis); opium poisoning.

Irregular, in last stages of valvular heart disease; myocarditis; profound anemia (on exertion); nervous palpitation; indigestion (flatulent colic).

In the irregular pulse we distinguish the :—

1. Intermittent pulse—
   Pulsus alternans (every second beat weak).
   Pulsus bigeminus (every third beat weak).
   Pulsus trigeminus (every fourth beat weak).
2. Intercidens pulse (several regular beats suddenly followed by a small beat and pause), in heart weakness.
3. Paradoxic pulse (the pulse grows smaller or ceases entirely on deep inspiration), in adhesive pericarditis; constriction of the air-passage; mediastinal tumors.

4. Dicrotic or double pulse (in part explained by a loss in the muscular tone in the arteries, so that the arterial impulse is separated from that of the ventricles by a perceptible interval), in typhoid fever and less marked in other acute febrile diseases; in chronic wasting diseases, especially tuberculosis; in anemias; after great loss of blood.

Asymmetric (radial pulse), in congenital anatomical variations of the artery on one side; acquired narrowing, compression, or cicatricial contraction of the radial, brachial, axillary, subclavian or innominate artery; aneurism of the aforementioned arteries or of the aorta; in pneumothorax compressing the subclavian artery.

THE ABDOMEN AND ITS CONTENTS.

In order to save time, inspection and palpation of the abdomen may at once be supplemented by percussion, succussion, etc. To judge matters correctly we should bear in mind the normal relations of the abdominal parietes to the underlying structures.

The **abdominal wall** is moderately arched; readily compressible without undue resistance or pain; moves slightly upward and downward quite evenly and regularly with inspiration and expiration; and on percussion yields a loud, tympanitic sound over all portions of the abdomen engaged by the intestines.

The **stomach** at birth is nearly cylindrical and lies obliquely in the abdominal cavity. Gradually the fundus increases in size and the stomach assumes a transverse position in such a manner that five-sixths of its volume occupies the left half of the abdomen and one-sixth the right. The capacity of the stomach varies, of course, with the age and size of the child, as fully given when discussing "infant feeding" (page 77).

The infantile **intestines,** especially the small intestine, are relatively longer than those of the adult. At birth the small intestine is about nine feet long, the large intestine about eighteen inches, the sigmoid flexure forming about half of the colon. The capacity of the infantile intestines is relatively greater than in the adult, but their musculature is thinner and weaker, hence the tendency to constipation and colic.

The liver of the newly born is relatively very large in size, much larger than in the adult, constituting in the former about one-eighteenth and in the latter about one thirty-sixth of the entire body weight.

As the child grows older the size of the liver is greatly reduced, but owing to the sloping course of the lower ribs the liver appears considerably larger than it actually is.

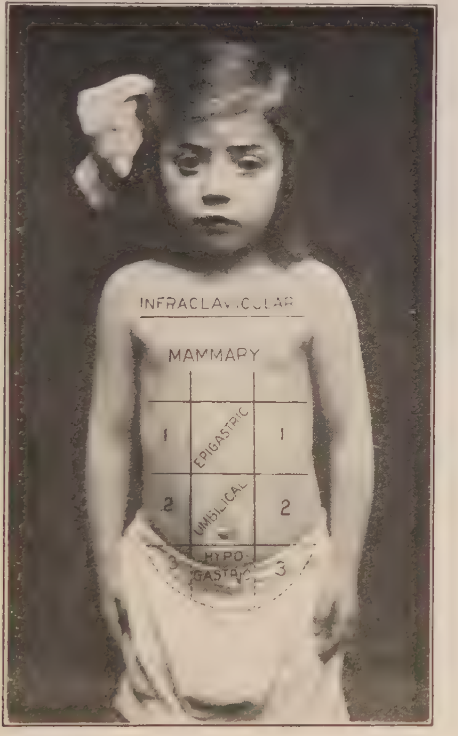

Fig. 17.—The Thoracic and Abdominal Regions. 1. Hypochondriac. 2. Lumbar. 3. Inguinal. (*Sheffield.*)

Normal **boundaries** of the liver (as determined by percussion):
Upper border, midsternal line, base of ensiform cartilage.
Mammary line, sixth rib.
Midaxillary line, eighth rib.
Scapular line, tenth rib.
Lower border, parasternal line, seventh rib.
Mammary line, about half an inch below free border of ribs.

Midaxillary line, tenth rib.

Scapular line, eleventh rib.

Left border, joins lower absolute heart-dullness.

Right border, joins the right kidney.

Its position varies greatly with the ascent and descent of the diaphragm—rises with expiration and descends with deep inspiration. In the same manner it rises with intestinal meteorism, and descends with overdistention of the lungs through disease, *e.g.*, emphysema or pneumothorax.

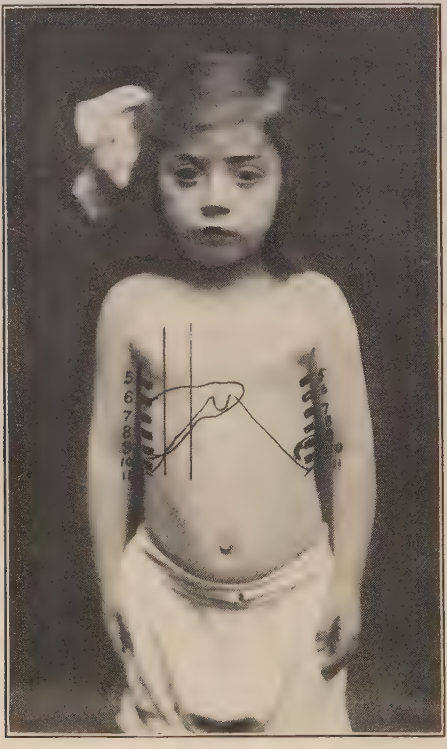

Fig. 18.—Topography of the Liver and Spleen. (*Sheffield.*)

The spleen lies in close contact with the diaphragm, and extends from the left midaxillary line to a point near the left border of the spinal column. Its upper border follows the ninth rib, its lower border the eleventh rib, for the most part bounding the left kidney. Normally the spleen cannot be outlined by percussion, but during deep inspiration it can sometimes be palpated at the free borders of the tenth and eleventh ribs.

The kidneys are situated upon the right and left sides of the spinal column, and extend from the levels of the twelfth dorsal to the second lumbar vertebræ. The uppermost end of the right kidney (the suprarenal capsule) is slightly overlapped by the liver and that of the left kidney by the spleen. Normal kidneys are occasionally palpable, but can never be outlined by percussion.

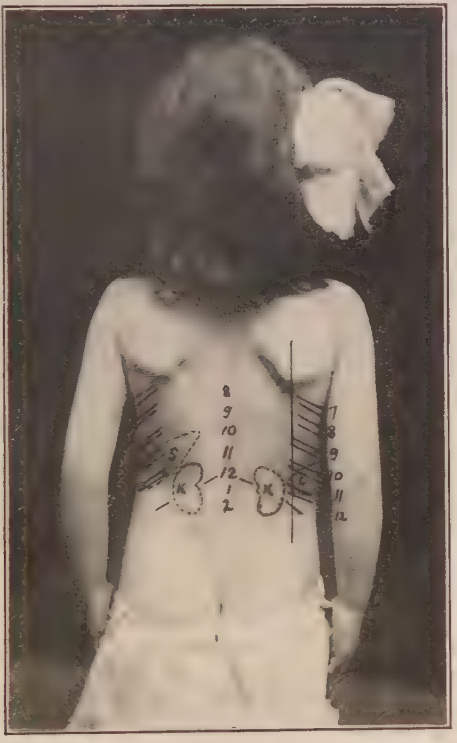

Fig. 19.—Topography of Kidneys, Spleen, and Liver. *S.* Spleen. *L.* Liver. *K.* Kidneys. (*Sheffield.*)

The urinary bladder is situated underneath the symphysis pubis, but when fully distended rises above it, eliciting dull percussion resonance.

Abnormal **size** and **shape** of **abdomen**—

Large and uniform, in flatulence; acute and chronic gastro-enteritis; acute peritonitis from various causes; intestinal atony or paralysis; extensive ascites.

Large and irregular, in *gastrointestinal* disease (congenital megacolon, pyloric stenosis, intussusception, appendical tumor or abscess, fecal impaction, strangulation, helminthiasis, tuberculosis); in *peritoneal* or *omental* affections (chronic peritonitis, tuberculosis, sacculated abscess, sarcoma, cysts); in *liver* disease (congestion, abscess, syphilis, rachitis, fatty or amyloid degeneration, leukemia, pseudoleukemia, hypertrophic cirrhosis, neoplasms); in *spleen* affections (leukemia, pseudoleukemia infantum, rickets, syphilis, typhoid, malaria, sepsis, neoplasms); in *kidney* disease (floating kidney, perinephritic abscess, neoplasms, hydronephrosis); also overdistention of the urinary *bladder;* large *ovarian* cysts; local injuries of the *abdominal wall.*

Retracted, in collapse especially from gastro-intestinal disease; in inanition (pyloric or esophageal stenosis); in meningitis ("scaphoid abdomen"); general cachexia and loss of fat and muscle.

omit! Increased **abdominal resistance**—

Local, in localized affections of the different abdominal organs (tumors, abscesses, foreign bodies, *e.g.*, fecal impaction; helminthiasis). worms in body

General, in hyperesthesia; rheumatism of abdominal muscles; colic; peritonitis from different causes; appendicitis; sclerema; scleredema; extensive dropsical effusion.

omit x **Abdominal pain**—

In all conditions enumerated under "abdominal resistance," except sclerema, scleredema, and dropsy. In pneumonia, pleurisy—reflex; in cholelithiasis; gastralgia; ulcer; nephrolithiasis; cystitis; vesical calculi; intestinal adhesions; ren mobilis; uterine and ovarian disease (in older girls); in hysteria.

omit **Visible intestinal peristalsis**—

Normal, in very thin and lax abdominal parietes, *e.g.*, congenital diastasis recti abdominis (see Fig. 37); infantile athrepsia; atrophy due to paralysis.

Abnormal (increased or reversed), in pylorus stenosis; intestinal obstruction or constriction from various causes; congenital dilatation of the colon.

Palpable or visible herniæ—

- In the linea alba (ventral; diastasis recti abdominis).
- At the umbilicus (congenital hernia of the cord—ectopia viscerum; simple umbilical hernia).
- In the lumbar triangles (lumbar hernia; lateral ventral hernia).
- In the inguinal regions (direct and oblique inguinal herniæ).
- At the femoral fossa (femoral or crural hernia).

Vomiting—

- Gastro-enteric (associated with nausea and effort; followed by relief); in simple gastroenteric disturbances and intoxication; pyloric stenosis or spasm; intestinal obstruction from various causes; appendicitis; peritonitis; the effect of emetics or poisonous drugs (taken by mouth).
- Cerebral (explosive; watery, recurrent without relief).
  - Direct, in acute and chronic affections of the cerebrospinal system; shock; psychic emotion.
  - Reflex, in extracranial irritation of the cranial nerves, *e.g.*, of the optic or oculomotor nerves in visual defects; of the auditory nerve, in otitides; pneumogastric, in pulmonary and cardiac diseases. Also in toxemia, by bacterial or chemical products (*e.g.*, sepsis, uremia, etc.). To the latter group belong also the so-called "cyclic" vomiting and the vomiting accompanying migraine.

Vomitus—

- Mucous, in chronic catarrh of the stomach; after swallowing large quantities of expectoration, in nasopharyngeal and laryngeal inflammation or pertussis.
- Bilious (yellowish-green or green), in gastro-enteric disturbances after repeated vomiting; in peritonitis; intestinal obstruction; liver affections.
- Bloody (hematemesis), in hemophilia and melena neonatorum; congenital obliteration of the bile-ducts; cirrhosis of the liver; ulceration of the lining of alimentary tract, especially of the upper part (from corrosive poisons; syphilis, etc); in vicarious menstruation.
- Purulent, in rupture into the stomach of large abscesses in the adjacent organs (*e.g.*, empyema).

Fecal, in severe intestinal obstruction with reversed peristalsis (*e.g.*, intussusception).

Parasitic, in helminthiasis; ankylostomum duodenale; trichinæ; echinococci.

Take

Diarrhea[1]—One to two movements in twenty-four hours are looked upon as normal. But even double the number of evacuations is not necessarily a manifestation of a pathologic condition unless the consistency, color and odor of the stools are materially altered. As on the first visit a specimen of the stool is not always obtainable, and if obtained not invariably of the same consistence as the preceding movements, it is important to gather all the information possible as to the abnormality in question—number, time of occurrence, quantity and quality.

1. Acute diarrhea occurs after the administration of cathartics or corrosives; in indigestion; stomatitis; gastroenterocolitis; proctitis and dysentery (blood, mucus and often pus); acute peritonitis; during the course of divers infectious diseases, especially cholera, typhoid, scarlatina, measles, influenza, sepsis, etc.
2. Chronic diarrhea is observed in dyspepsia; chronic gastroenterocolitis; chronic proctitis and dysentery (amebic); intestinal tuberculosis and other chronic wasting diseases (especially syphilis, leukemia, amyloidosis); helminthiasis (especially in trichocephalus and ankylostomum—often mucosanguinolent stools); malaria (periodic); intestinal lithiasis (mucus, blood and sand), and in partial intestinal stenosis (band-like, flat, mixed with mucus).

Constipation[2]—In determining the clinical significance of constipation, inquiry should be made as regards the duration of the constipation, mode of feeding of the child, presence or absence of vomiting and tenesmus, and the color and consistency of the stools.

1. Habitual constipation occurs in consequence of insufficient (pyloric stenosis) or improper feeding (lack of fat, water, etc., excess of starches, etc.); intestinal atony (from a great number of causes, *e.g.*, congenital or acquired muscular insufficiency—megacolon, or

---

[1] See "Infants' Stools," page 43.

[2] *Ibid.*

artificial distention), general debility, cretinism, etc.; partial intestinal obstruction (*e.g.*, hernia, neoplasms) and abstinence owing to painful lesions in the rectum (*e.g.*, hemorrhoids, fissures).

2. Acute constipation, with persistent vomiting, pain, meteorism, etc.—in all forms of congenital intestinal atresia and acquired acute intestinal obstruction (intussusception, strangulation, fecal impaction, peritonitis, appendicitis, and volvulus).

Take — x

INFANTS' STOOLS.

The character (consistency, color, reaction, odor, etc.) of infants' stools greatly depends upon the kind and quantity of food consumed.

Normal stools are—

- Soft and pasty, golden yellow, slightly acid and almost odorless, in breast-milk feeding.
- Soft—putty-like—whitish-yellow, slightly alkaline and slightly offensive in odor, in cows' milk feeding.
- Soft—salve-like—yellowish-brown or brown, slightly alkaline or neutral, and malt-like in odor, in feeding with malted or farinaceous foods.

Abnormal Stools—

(*a*) Consistency—

1 Thick and formed, in deficiency of fat supply; excess of starches; habitual constipation.

2 Soft, smeary, like moistened shavings of soap, in fat indigestion.

3 Soft or hard and mixed with tough white curds, in casein indigestion.

4 Thin, watery, in catarrhal gastroenteritis; typhoid fever; from the effects of hydragogue cathartics; rectal stricture (*e.g.*, syphilitic).

Serous, in severe gastroenterocolitis; cholera.

Mucous, in obstinate constipation with tenesmus; in disease of the large intestine (large quantity); in disease of small intestine (mixed with feces).

Bloody, in rectal affections (*e.g.*, proctitis, hemorrhoids, fissure, polypus, prolapsus); dysentery; intussusception; hemorrhagic disease (*e.g.*, melena, purpura, hemophilia, etc.); foreign body in rectum. [illegible]

(*b*) Color—
- Yellowish-green, in gastrointestinal indigestion (especially of casein).
- Green, in gastroenteritis; from the effects of calomel.
- Clay-color, in obstruction to the flow of bile.
- Black, in meconium; from the effects of iron, manganese and bismuth; also blood (coming from upper portion of the bowels).
- Red, from admixture of blood (from lower portion of bowels, especially rectum).

(*c*) Reaction—
- Decidedly alkaline, in proteid indigestion.
- Moderately acid, in fat indigestion (from fatty acids); carbohydrate indigestion (acetic or lactic acid).

(*d*) Odor—
- Foul, in proteid indigestion; fermentation.
- Rancid, in fat indigestion.
- Sour or pungent, in carbohydrate indigestion.

The stools should be examined also for parasites (see "Intestinal Worms" page *222*) and calculi.

PRINCIPAL ABNORMALITIES OF URINE.

In male infants the urine may be collected by placing the penis in a test-tube or the neck of small bottle fastened by means of strips of adhesive plaster; in female infants, by placing absorbent cotton in front of the vulva. Where these measures fail, catheterization should be resorted to.

Traces of albumin and sugar; occasionally hyaline and granular casts; a moderate amount of mucus, uric acid crystals and urea, are found in the urine of healthy infants a few weeks' old.

The quantity of urine passed in twenty-four hours is larger in infants than in older children, but varies with the amount of liquid consumed. It is smaller in breast-fed than in bottle-fed babies.

Polyuria (increased amount of urine)—
- Diabetes mellitus.
- Diabetes insipidus.
- Contracted kidney.
- Granular atrophy of the kidney.

- Amyloid kidney.
- Convalescence after acute diseases (epicritic polyuria).
- Disease of the nervous system, functional and organic, as hysteria, neurasthenia, migraine, chorea, epilepsy, tabes, cerebrospinal meningitis.
- Medicinal (acetates, salicylates, digitalis, calomel, etc.).

Oliguria (decreased secretion of urine)—

- Febrile conditions.
- Profuse diarrhea.
- Circulatory disturbances.
- Acute nephritis.
- Some forms of chronic nephritis.

Anuria (suppression of urine)—

- Uremia.
- Acute anemia.
- Catarrh of the stomach or intestines.
- Cholera.
- Dysentery.
- Nervous manifestations.
- Lead colic.
- Poisoning with arsenic, corrosive sublimate, morphine, atropine, oxalic acid, etc.

Glycosuria—Sugar -

- Constant in diabetes mellitus.
- Transient glycosuria—
  - Cholera.
  - Typhoid fever.
  - Intermittent fever, particularly during convalescence.
  - Syphilis.
  - Scarlatina.
  - Measles.
  - Diphtheria.
  - Influenza.
  - Gout.
  - Disease of the lungs and liver.
  - Disease of the brain, especially if involving the fourth ventricle.
  - Cerebrospinal meningitis.
  - Tetanus.
  - Lesions affecting the central and peripheral nervous system.

- Poisoning with morphine, atropine, strychnine, oxalic acid, carbon monoxide, lead, chromates, chloroform, ether, etc.
- Transient alimentary glycosuria—
  - Disorder of the stomach.
  - Overingestion of starchy and saccharine foods.
  - Cirrhosis of the liver.
  - Morbus Basedowii.
  - Disease of the heart.
  - Phosphorus poisoning.
  - Atrophy of the liver.
  - Traumatic neuroses.
  - Fatty degeneration of the liver.
  - Psoriasis.

Aceton—

- Diabetes mellitus, especially in advanced cases; diabetic coma.
- Fever.
- Carcinoma.
- Auto-intoxication.
- Psychoses.
- After chloroform narcosis.

Diacetic acid—

- Diabetes mellitus, advanced cases.
- Auto-intoxication (diaceturia).

Albuminuria—

- (*a*) Renal (nephritis, pyelitis, pyelonephritis, nephrolithiasis) and vesical (calculi, colicystitis).
- (*b*) Changes in the constitution of the blood:
  - Ischemia.
  - Anemia.
  - Struma.
  - General weakness.
  - Effect of certain poisons, as cantharides, mustard, oil of turpentine, carbolic acid, alcohol, lead, etc.
  - Infectious fevers—micro-organisms in the blood.
  - Febrile conditions.
- (*c*) Disturbance in the circulation:
  - Acceleration of the arterial current.
  - Slowing of the venous current.
  - Prolonged muscular exercise.

After cold baths.
After epileptic fits.
Compression of the thorax.
Derangement of the cerebrospinal system.
(*d*) Functional.
Orthotic.
(*e*) Digestive.
Ingestion of excessive quantities of albumin (*e.g.*, eggs, cheese, raw beef).

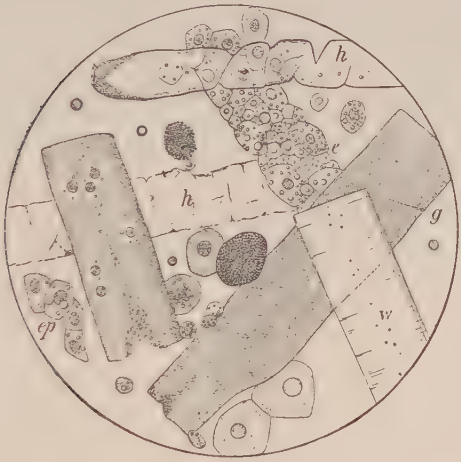

Fig. 20.—Severe Acute (at first decidedly hemorrhagic) Nephritis, which Ended Fatally in Four Weeks. × 350. *h*, Hyaline cast. *g*, Granular cast. *w*, Waxy cast. *e*, Epithelial cast. *ep*, Free renal epithelia. Also two finely granular, uniformly fatty renal epithelia. (*Lenhartz.*)

Casts—
Hyaline (narrow and broad):
  Acute and chronic nephritis.
Granular (coarse and fine granules):
  Chronic pathologic conditions of the kidney.
Epithelial:
  Inflammation in the anatomical structure.
Bloody:
  Hematuria.
  Acute diffuse nephritis.
  Acute renal congestion.
  Hemorrhagic infarction of the kidney.

Fatty:
Fatty changes in the kidney, large white kidney.
Waxy:
Amyloid kidney and many forms of nephritis.
Bacterial:
Interstitial suppurative nephritis, ascending pyelonephritis.
Purulent:
Abscess of the kidney.

Uric acid (pathologic when deposit occurs shortly after urine is voided)—
Acute fevers.
Inflammation.
Increased tissue metabolism.
Defective physiologic action of the liver.
Sedentary habits of life.
Early stages of interstitial nephritis.
Convalescence from scarlatina.

Hematuria (blood)—
(*a*) Renal:
Bright's disease.
Amyloid disease.
Malignant growths.
Tuberculosis.
Renal calculi.
Cystic disease of the kidney.
Abscess.
Renal embolism.
Hydatids.
Acute febrile processes.
Purpura hemorrhagica.
Traumatism involving the kidney.
Medicinal, as turpentine, cantharides, arsenic, etc.
(*b*) Vesical:
Stone in the bladder.
Cystitis.
Neoplasms of the bladder.
(*c*) Urethral:
Acute gonorrhea.
Neoplasms.
Traumatism.

Pyuria (pus)—
(*a*) Renal:
Pyelonephritis.
Pyelitis.
Cancer.
Tuberculosis.
Nephritic abscess.
(*b*) Vesical:
Cystitis.
Vesical stone.
Ulceration.
Tuberculosis.
(*c*) Urethral:
Gonorrhea.
Rupture of abscess in urinary passages.
Urethritis.

Peptonuria (pepton)—
Croupous pneumonia.
Bronchopneumonia.
Empyema.
Phthisis pulmonum.
Typhoid fever.
Scarlet fever.
Malaria.
Erysipelas.
Epidemic cerebrospinal meningitis.
Purpura hemorrhagica—diverse forms.
Scurvy.

Bacteriuria (pathogenic)—
Gonococcus.
Tubercle bacillus.
Colon bacillus.
Strepto- or staphylo-cocci.

Parasituria—
Distomum hæmatobium.
Hooklets of echinococcus.
Filaria.

THE GENITALIA.

In the male child we should look for abnormalities of the penis (malformations, adhesions of the prepuce, phimosis, overstretched prepuce,—masturbation,—faulty location of the urethral orifice, urethral discharge), scrotum and its contents (tumefactions, undescended testicles).

Scrotal tumefactions—
Communicating with abdominal cavity, in hernia; hydrocele; and, higher up in the inguinal canal, partly descended testicle.
Non-communicating with abdominal wall, in orchitis (not rarely with parotitis); epididymitis; syphilis, tuberculosis, cysts, and malignant growths of testicle.
Dropsical effusions, of renal or cardiac origin or edema from circulatory disturbance in the spermatic cord.
Local scrotal inflammation, in abscess; erysipelas; gangrene; sebaceous cysts; traumatism.

In the female we should note the presence of labial hernia or hematoma, vaginal discharges or deposits (in diphtheria, noma); enlarged clitoris or preputial adhesions; atresia vaginæ; abnormalities of the hymen (imperforate).

Vulvovaginal discharge—
Mucous, white, in simple catarrhal vulvovaginitis (from lack of cleanliness; irritating urine).
Purulent, yellow or yellowish-green, in gonorrheal vulvo-

vaginitis or infection by other micro-organisms (*e.g.*, streptococcus in exanthematous diseases).

Hemorrhagic, in hemorrhagic diathesis (in the newly-born and in older children); in vulvovaginitis with erosions of the mucous membrane (sometimes after severe local treatment); prolapse of the urethra; neoplasms; menstruatio præcox.

Take —

THE RECTUM.

Abnormalities of the rectum can readily be detected by inspection (sometimes with the aid of proctoscope) and digital examination. We should look for condylomata, fistulæ, hemorrhoids, polyps, prolapse, intussuscepted intestine, fissures, pinworms, foreign bodies and discharges.

Rectal discharges—

Mucous, mucopurulent, and slightly bloody, in simple proctitis; rectal fissure or fistula; colitis.

Purulent, in communicating ischiorectal abscess; gonorrheal proctitis; impacted foreign body.

Hemorrhagic, in hemorrhoids; polyps; dysentery; ulcerative proctitis (tuberculous, or otherwise); intussusception; prolapsus recti; hemorrhagic diathesis.

Take —

THE VERTEBRAL COLUMN.

The vertebral column of the infant under six months is quite straight, except for a slight dorsal curve. As the child grows older and attains the power of sitting, standing and walking, we soon find the dorsal region of the spinal column curved posteriorly and the cervical and lumbar regions anteriorly—compensatory curvatures. At first these curves disappear in recumbent posture, but they become permanent at about the age of six. The normal spinal column is perfectly movable.

In the physical examination of the spinal column we note the presence of:—

Deformities (lordosis, kyphosis and scoliosis)—

Congenital, in osteogenesis imperfecta, etc., cervical rib.

Habitual, or postural from faulty posture; the effect of superencumbrance (carrying of heavy weights upon the back or shoulders).

- Static, the result of oblique pelvis, *e.g.*, congenital or acquired shortening of one lower extremity in hip-joint disease.
- Tuberculous, in vertebral caries.
- Neuromuscular, in muscular insufficiency (to which belongs also rachitic deformity of the spine), or paralysis, *e.g.*, poliomyelitis; pseudoparalysis.

Clefts, usually congenital, *e.g.*, spina bifida.

Protrusion [illegible] of [illegible] cord –

Tumors—

tumor containing [illegible]

- Congenital, in teratomas; hernial protrusions.
- Acquired, in vertebral caries, osteoma.

Stiffness (with or without pain)—

- Central, in meningitis; meningeal irritation (*e.g.*, apex pneumonia; hydrocephaloid).
- Spinal, in disease of the spinal cord (*e.g.*, spinal meningitis, myelitis); in trauma or disease of the vertebræ or articulation (*e.g.*, vertebral caries, spondylarthritis). Also cervical rib; osteoma.
- Neuromuscular, in neuralgia; myalgia; myositis..

For further information the reader is referred to "Attitude of the Head and Neck" and "Spondylitis."

Take –

THE EXTREMITIES.

The extremities should be examined with a great deal of care—inspected, measured, palpated, percussed—as their anomalies in form and disturbances in function, etc., furnish most instructive information not alone as to existence of local disease, but also of general systemic affections, pre-eminently those of the nerve system.

Shortness of—

- Single limbs, in paralytic, hysterical or traumatic (*e.g.*, after fracture) contractures; hip-joint disease; congenital deformities.
- All extremities, in achondroplasia (as compared with the long trunk).

Curvatures—

- Congenital, in diverse congenital malformations (*e.g.*, osteogenesis imperfecta; osteomalacia; achondroplasia).
- Acquired, after fractures; in syphilis; rachitis; tuberculosis.

Tumefactions—

- Diaphyseal, tuberculous and non-tuberculous in periostitis;

osteitis; osteomyelitis; syphilis; exostosis; malignant growths; after fracture.

Epiphyseal, the same as in diaphyseal, also in rachitis; Barlow's disease; arthritis deformans; rheumatic affections; septic arthritides; hemarthrosis (hemophilia, purpura peliosis rheumatica); synovitis; bursitis; "intermittent hydrops."

Muscular Weakness, "flaccidity" (with or without atrophy):

Without True Paralysis, in pseudoparalysis of syphilitic origin (upper extremities); Barlow's disease; osteomyelitis; osteomalacia; polyarthritis and myositis; traumatism to the muscles or bones (dislocation or fracture); progressive muscular atrophies (muscular and neurospinal types); idiocy (especially amaurotic family idiocy) and cretinism; rachitis and muscular debility after prolonged sickness (in bed); hysteria.

With Paresis or Paralysis, in poliomyelitis (early); myelitis (the muscular involvement depending upon the seat of the lesion in the cord) Landry's paralysis; spinal meningitis (chronic); polyneuritis (usually bilateral and symmetrical) from various causes; birth palsies.

Muscular Contracture, "spasticity" (with or without atrophy):

Without True Paralysis, in trismus and tetanus traumaticus and neonatorum; meningismus; early stage of meningitis; tetany; pseudotetanus; tetanism (*q. v.*); eclampsia infantilis; mus. spasm myotonia; catalepsy; hysteria trichinosis; hydrocephaloid.

With Paresis or Paralysis, in all forms of cerebral paralysis (cerebral hemorrhage, embolism, abscess, tumor, sclerosis, tuberculosis, encephalitis, porencephalia, hydro- or micro-cephalus, etc.); myelitis (late stage); spastic spinal paralysis; Little's disease; amyotrophic lateral sclerosis.

Spasmodic Movements (see also "Convulsions"):

Intention tremor, in disseminated sclerosis; ataxia hereditaria; spastic spinal paralysis; myotonia congenita.

Irregular shaking, in cerebral hemorrhage; tumor; encephalitis; hydrocephalus; all forms of meningitis; toxic neuritis, especially diphtheritic and uremic; hysteria; Jacksonian epilepsy; idiocy.

Fibrillary twitching, in progressive muscular atrophy; acute febrile diseases; neuroses; strychnin poisoning.

Athetoid movements, in chronic brain affections, especially of the internal capsule.

Choreiform movements, in all forms of chorea; spastic cerebral paralysis; paramyoclonus multiplex; hysteria; tic.

Paralysis—

Unilateral.

Upper and lower, in lesions of one cerebral hemisphere, *e.g.*, cerebral hemorrhage, embolism, abscess, tumor, sclerosis, encephalitis, meningitis, depressed fracture, porencephalia, etc.; poliomyelitis.

Upper, in unilateral, cerebral lesion of the arm center, *e.g.*, embolism, tubercle, etc.; unilateral spinal lesion of the cervical region, *e.g.*, incipient spondylitis, etc.; traumatism to the brachial plexus, *e.g.*, birth palsy; poliomyelitis; regressive stage after hemiplegia.

Lower, in unilateral cerebral lesion of the leg center (same as in upper); unilateral spinal lesion in the lumbar region; trauma of the lumbar plexus; poliomyelitis.

Bilateral.

Upper and lower, in bilateral lesions of the brain (cortex, pons, or medulla), *e.g.*, intracranial hemorrhage, multiple growths, especially tuberculous and syphilitic, disseminated sclerosis, etc., spinal sclerosis; spinal meningitis; poliomyelitis; Landry's paralysis (late); progressive muscular atrophy (late); amyotrophic lateral sclerosis (late); syringomyelia (late); multiple neuritis; amaurotic family idiocy (late).

faulty dev.

Upper, in double trauma of the brachial plexus or individual cords, *e.g.*, compression in instrumental delivery; transverse cervical myelitis; poliomyelitis; Landry's paralysis (late); bilateral cerebral lesions of the arm centers; syringomyelia (early).

Lower, in bilateral trauma of the lumbar plexus or its main branches; transverse lumbar myelitis; transverse dorsal myelitis (late); spastic spinal paralysis;

hereditary ataxia (late); tabes dorsalis (late); polyneuritis, especially diphtheritic (early); amyotrophic lateral sclerosis (early); poliomyelitis; bilateral cerebral lesions of the leg centers; hydrocephalus.

Localized paralysis of principal muscles concerned in movements of the extremities and their nerve supply.

- Upper extremities.
  - Trapezius (spinal accessory nerve): sinking of shoulder downward and forward; rotation of scapula outward and upward; elevation of shoulder imperfect.
  - Serratus magnus (long thoracic nerve): slight rotation of scapula; difficulty of raising arm above shoulder; deep furrow between scapula and vertebræ on moving arm upward.
  - Pectorales (anterior thoracic nerves): impaired abduction of upper arm; placing of affected hand on healthy shoulder impossible.
  - Teres major and subscapular (subscapular nerves): Loss of inward rotation of arm.
  - Infraspinatus (suprascapular nerve) and teres minor (axillary nerve): loss of outward rotation of arm.
  - Latissimus dorsi (subscapular nerve): impaired abduction of arm; inability to place hand on sacrum.
  - Deltoid (circumflex nerve): inability to elevate arm; atrophy.
  - Biceps and brachialis anticus (musculocutaneous): inability to flex forearm, when in supination; inability to supinate forearm, when flexed.
  - Supinator longus and brevis (musculospiral nerve): weakened flexion when forearm is half-pronated; inability to supinate with the forearm extended and pronated.
  - Triceps and the extensors (musculospiral nerve, "radial paralysis"): inability to extend forearm (in triceps paralysis); hand-drop in flexed position; flexion of fingers; impaired abduction

and adduction (paralysis of the extensors); impaired sensation along radial side; atrophy.

Flexor carpi ulnaris, profundus digitorum, minimi digiti, and inner head of brevis pollicis; the interossei, lumbricalis, palmaris brevis (ulnar nerve, "ulnar paralysis"): claw-like deformity of hand.

Pronator radii teres, pronator quadratus, palmaris longus; flexors carpi radialis, sublimis digitorum, profundus digitorum, and longus pollicis (median nerve, "median paralysis"): abolition of power of pronation; inability to flex terminal phalanges and thumb; objects can be grasped with the last three fingers only; trophic and sensory disturbance.

See also "Birth-palsy."

Lower extremities.

Gluteus maximus and minimus (gluteal nerve): difficulty to abduct thigh; to walk up-hill; to rise from sitting posture; impairment of circumduction and inward rotation, and walking; toes are turned inward.

Anterior muscles of thigh, except tensor vaginæ femoris (anterior crural nerve, "crural paralysis"): inability to flex thigh on trunk and to flex trunk when in recumbent posture; to extend leg when flexed; difficulty to stand or walk, or to rise from kneeling posture.

Obturator externus and the abductors (obturator nerve): impaired adduction and outward rotation of thigh; inability to cross legs.

Biceps, semimembranosus, semitendinosus, the flexors of knee (great sciatic nerve): inability to flex knee; difficult locomotion; leg inverted or everted.

Gastrocnemius, soleus, and plantaris—the extensors of the foot (internal popliteal nerve): inability to extend (plantar flexion) of foot; to stand on tip-toe; difficulty in walking; foot everted, ankle lowered (talipes calcaneus).

Peroneus longus (musculocutaneous): foot inverted; plantar arch flattened (flat-foot).

Tibialis anticus, and extensor longus digitorum—flexors of foot (anterior tibial nerve): impaired flexion, abduction and adduction (talipes equinus).

Peroneus brevis, and tibialis posticus (posterior tibial nerve): inability to adduct or abduct foot without flexion or extension. Talipes valgus in tibial paralysis; talipes varus in peroneal paralysis.

Peculiarities of gait—

Dragging, in multiple sclerosis; spastic spinal paralysis; poliomyelitis involving both legs; amyotrophic lateral sclerosis; hemiplegia, and cretinism.

Straddling, in tabes dorsalis.

Staggering, reeling, in multiple neuritis; hereditary ataxia; cerebellar disease.

Waddling, in progressive muscular dystrophy; bilateral dislocation of the hips; rachitis.

Hobbling, in osteomalacia.

Shuffling, in hysterical paralysis.

Tendon reflexes—

Knee-jerk.[1]

Exaggerated, in spinal or cerebral paralysis, associated with "spasticity" of the muscles (see page 52); also in transverse myelitis affecting the spinal cord above the second lumbar vertebra; cerebellar disease; general nervousness.

Diminished or lost, in spinal or neural affections associated with "flaccidity" of the musculature (see page 52); also in transverse myelitis below the second or third lumbar vertebra; hereditary ataxia; "meningismus" (early stage).

Ankle clonus.[2]

Absent or very slight, in good health.

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[1] Obtained by a sharp blow over ligamentum patellæ, while lower leg hangs loosely down.

[2] Rhythmic oscillation of the foot, elicited by abruptly pressing toes upward with one hand, while supporting the leg with the other hand.

Present, and often very pronounced, in cerebral hemorrhage; spastic spinal paralysis; dorsal myelitis; disseminated lateral sclerosis; hysterical paralysis; tetanus.

Periosteal reflex.[3]

Slight, in good health.

Greatly exaggerated, in cerebral hemorrhage.

Kernig's sign (inability to extend legs when thighs are flexed on abdomen): in divers forms of meningitis; occasionally in typhoid fever.

Babinski's reflex (extension of great toe with flexion of other toes on touching sole of foot): pathognomonic of meningitis in children over two years of age; in organic hemiplegia.

Table – WEIGHT AND LENGTH OF A CHILD.

An authentic record of the gain or loss in weight of the patient is invaluable in the diagnosis, prognosis and treatment. There is no absolute standard for the normal weight or height of an infant or older child. To a great extent it depends upon the race the child descends from and also upon family disposition. Furthermore the size of the child is not always an indication as to his inherent vigor. Ordinarily boys are heavier than girls.

[illegible]

TABLE OF COMPARATIVE WEIGHT AND HEIGHT OF A NORMAL CHILD.

AGE.	LENGTH.	WEIGHT.	AGE.	LENGTH.	WEIGHT.
Birth.	19.5 Inches.	7 Pounds.	2 Years.	32 Inches.	26 Pounds.
1 Month.	20.5 “	7¾ “	3 “	35 “	31 “
2 Months.	21. “	9½ “	4 “	38 “	35 “
3 “	22. “	11 “	5 “	41 “	40 “
4 “	23. “	12½ “	6 “	44 “	45 “
5 “	23.5 “	14 “	7 “	46 “	49 “
6 “	24. “	15 “	8 “	48 “	54 “
7 “	24.5 “	16 “	9 “	50 “	60 “
8 “	25. “	17 “	10 “	52 “	65 “
9 “	25.5 “	18 “	11 “	54 “	71 “
10 “	26. “	19 “	12 “	56 “	80 “
11 “	26.5 “	20 “			
12 “	27. “	21 “			

Weight is—

Diminished, rapidly, in cholera infantum; acute febrile diseases; chronic wasting diseases, especially tuberculosis, malignant growths and suppurative processes; in diabetes.

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[3] Jerk of hand or forearm produced by a tap upon the tendons of the supinator longus and biceps—at lower end of the radius and ulna; or of the triceps tendon at the olecranon.

Slowly, in dyspepsia; organic affections with slow course, *e.g.*, heart and kidney diseases.

Increased, rapidly, in adipositas, cretinism, anasarca. Slowly in normal health.

Height is—

At a standstill, in infantilism, cretinism, severe rachitis, and in marked central paralysis.

Increased, rapidly, in acute febrile diseases, especially typhoid fever. Slowly, in normal growth.

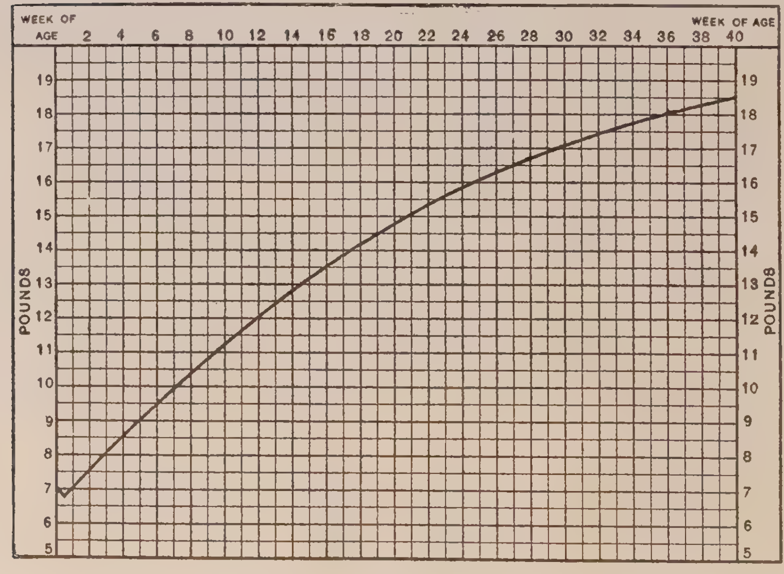

Fig. 21.—Weight Chart.

CHAPTER II.

Prevention and Control of Disease.

The warfare between health and disease.

THE warfare between health and disease evolves with inception of life of the organism. The battle is fiercely rampant and everlasting, the victory at best but temporary. Supremacy of health over disease fluctuates with the amount of inherent strength of the individual, the natural and acquired power of resistance, and the assistance received through prophylaxis and therapeusis.

Survival of the strongest.

Nature aims to exterminate the weak, and right at birth tests the vitality of the infant in a manner most hazardous to its subsistence. Thus, accustomed to the ideal domicile of the maternal uterus—protected from traumatism and atmospheric vicissitudes, nurtured without effort and animated without the touches of pain or distress—the newborn is suddenly cast upon its own resources into a sphere of eternal strife, where every organism, every element, is struggling for supremacy, and where the strongest—not invariably the fittest—triumphs.

INHERENT STRENGTH.

Fostering of inherent strength.

Inherent strength is essential to active life, to maintenance of perfect health. A powerful constitution will overcome an attack of disease that will fell the weak and the frail. A strong organization will surmount hardships and rapidly recuperate after protracted illness. Inherent strength is not procurable after birth. It is a consummation, an inheritance, of ancestral virility and vigor, premarital purity, conjugal devotion, matrimonial chastity, sobriety and ideal hygiene. It can be fostered by regulation of marriage, conservative mutual selection, prohibition of consanguineous marriages and those encumbered by grave disease, habits, alcoholism and drug addictions, or extreme poverty. Finally, it can be greatly improved by judicious management of pregnancy.

POWER OF RESISTANCE AND SUSCEPTIBILITY.

Natural and acquired.

Immunity, protection, or power of resistance against disease, and to a slighter extent also susceptibility toward disease, may be natural or acquired. It varies in different individuals and in the same individual at different periods of life. Natural or congenital immunity is aptly exemplified by the comparatively rare occurrence of communicable diseases in infants under three months of age. Congenital susceptibility is demonstrable by the prevalence of certain affections in some families or races, *e.g.*, hemophilia, amaurotic family idiocy and the like. In contrast to inherent vitality, acquired power of resistance is vastly influenced during the life of the child. Thus, immunity against communicable diseases is often temporarily or permanently conferred, naturally by a previous attack of the same malady (*e.g.*, yellow fever, pertussis), and artificially by: 1. Suitable nutrition. 2. Hygiene and sanitation. 3. Immunization. 4. Drugs and physical therapeutic measures.

I. NUTRITION.

Elements of nutrition.

Suitable nutrition is indispensable to the life and growth of the individual and to the maintenance and advancement of his power of resistance. The human economy demands for its sustenance a liberal supply of proteids (to build up and to reconstruct the tissues), fat and carbohydrates (to produce energy and heat), mineral salts (to help formation of bones and teeth), and water (to aid the solubility of the food elements and the excretion of waste products). An ideal food, therefore, must contain these five ingredients in more or less definite proportion, must be readily digestible and assimilable, and be free from pathogenic bacteria.

WOMAN'S MILK.[1]

Nature's infant food.

Woman's milk is a highly nutritious, biologically as yet somewhat mysterious product, destined by nature to serve as the food-supply alike for the rich and the poor, the weak and the strong infant under nine months of age. It not only complies with the aforementioned qualitative requisites, but being ready for immediate consumption—without previous pasteurization, sterilization or modification—at all hours of the day and at all seasons of the year, it is also the most convenient and satisfactory food from an economic point of view. Infants reared on woman's milk are almost invariably healthier, stronger and less troublesome than those that are bottle fed.

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[1] For its approximate composition, see footnote, page 68.

Ever ready.

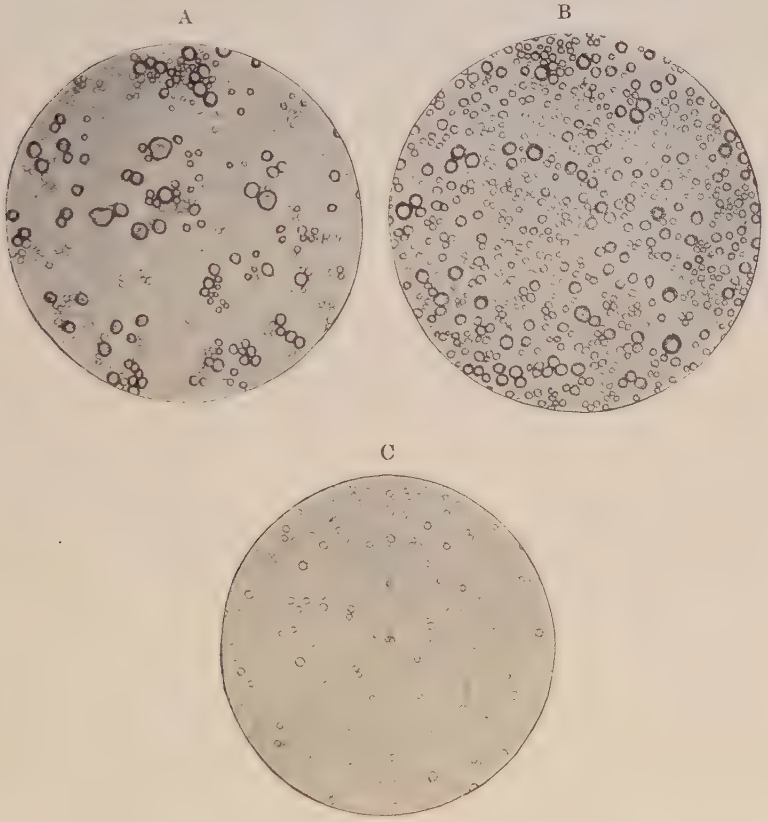

Fig. 22.—Microscopical Appearances of Woman's Milk. (After *Fleischman.*) *A,* Poor milk, showing preponderance of large fat globules and a paucity of fat. *B,* Normal milk, showing the preponderance of medium-sized fat globules. *C,* Poor milk; a paucity of fat and an almost granular state of the fat globules.

With suitable management they are, as a rule, free from gastroenteric affections, scurvy and rickets, and present greater power of resistance to communicable diseases.

MATERNAL NURSING.

For the reasons just given, and in view of the facts that wet-nurses are expensive luxuries, are often unreliable, and may at some time during the nursing period, through unscrupulous and impure contact, contract and convey a disease to her charge, it is the solemn duty of every healthy mother to endeavor to nurse her offspring, wholly or partially, even if it be only for a brief period of time.

Mother's duty.

Successful maternal nursing presupposes, in addition to general good health of the mother, well-developed breasts and nipples and an ample supply of rich milk. These qualifications are rarely met to perfection with women of large cities, where the extravagancies of extreme wealth or the misery of extreme poverty sap their vital forces. A great deal, however, can be accomplished by judicious management of the mother during pregnancy and parturition.

Prerequisites.

The prospective mother should be placed in the most healthful physical and mental condition. Her diet should be liberal, her living rooms spacious and airy and her surroundings cheerful. She is to be free from anxieties of a livelihood and the pompous frivolities of wanton society. The primipara should be taught to realize that pregnancy and parturition are physiologic processes, ordinarily devoid of perilous complications or sequelæ.

Care of the prospective mother.

Toward the end of pregnancy the breast nipples should be elongated by gentle traction with the fingers or pump, and cleansed and hardened by means of hot boric acid solutions, cognac, glycerite of tannin, and the like. To insure an ample supply of breast milk after delivery, in addition to complying with the aforementioned suggestions, a liberal fluid diet consisting principally of rich cows' milk, cornmeal and oatmeal gruel cooked in milk, malted milk, etc., forms the most efficient adjuvant. At a later period the dietary of the nursing mother should be increased by a liberal allowance of meat, eggs, vegetables and other nutritious food-stuffs to which she was ordinarily accustomed.

Liberal fluid diet during nursing.

Light outdoor exercise, regulation of the bowels, avoidance of fatigue and nerve disturbances, all serve well to improve the health of the mother and the quality of her milk, and indirectly the welfare of the baby.

One other special advantage of maternal- over wet-nursing is the benefit the newborn derives from the consumption of the provisional milk secretion—the colostrum. This deep yellow, strongly alkaline and albuminous fluid which forms the mammary secretion during the first three or four days after labor, not only acts as a laxative—which is badly needed—but being small in quantity it also serves to moderate the greedy appetite of the infant and prevents early overfeeding, the usual cause of infantile colic.

Colostrum.

The nursing of the baby is generally begun with about eight hours after delivery, or later if the mother has not fully recovered from the painful and fatiguing ordeal. During the first few days the infant is applied to the breast every three or four hours and afterward every two and a half hours. It should not be awakened for a feeding if sound asleep (except when very weak and delicate), and unless very restless should be left alone from 10 P.M. to 5 A.M. It should be nursed from fifteen to twenty minutes at a time, alternately on one and the other breast, or on both breasts if the milk secretion is scanty. From six weeks on the infant should be fed every three hours, and less frequently when it reaches six months of age. Between nursing the baby may receive a few ounces of warm, slightly sweetened water.

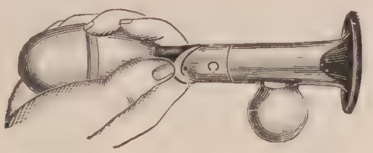

Fig. 23.—Breast Pump.

Time for nursing.

Before and after each feeding the breast nipples should be carefully cleansed with a warm saturated solution of boric acid.

If the breast nipples are short, sunken or cracked, we must temporarily resort to an artificial nipple or breast pump (see Fig. 23). The latter device is also employed where the infant is too weak to pull, or refuses to make an effort to do so. In very delicate infants, *e.g.*, prematures, it is often necessary to

Use of breast pump.

withdraw the breast milk with a pump[1] and administer it by means of a spoon or dropper.

With the suggestions here offered the majority of healthy mothers will be able to nurse their offspring, provided they are sufficiently encouraged by the physician and the enormous advantages of maternal breast feeding are thoroughly explained to them.

When an infant does not thrive on breast milk, it is imperative, before resorting to another infant food, to carefully analyze the breast milk, and, if possible, to overcome the difficulty. We should determine:—

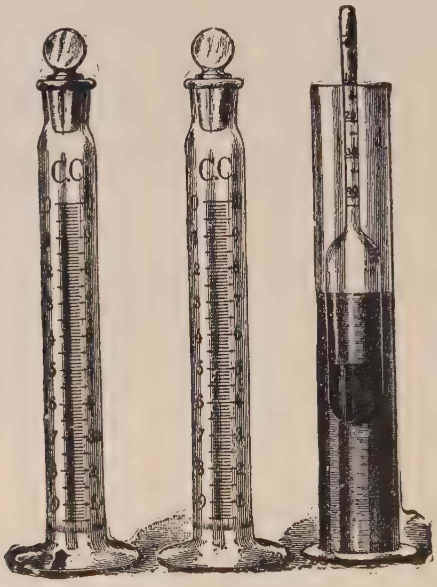

Fig. 24.—Holt's Milk Set.

Analysis of breast milk.

1. The *quantity*.—This can readily be learned by extracting the milk-supply of one or both breasts, or by weighing the infant before and after nursing and noting the difference in weight.

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[1] A practical method of withdrawing breast-milk is the following: Fill a wide-mouthed bottle with boiling water. Then pour the water out and cool the mouth of the bottle and apply over the nipple. As the bottle cools, the milk spurts from the nipple and the breast is automatically emptied. The milk is thus ready for use at once, keeping warm in the warm bottle.

2. The *quality*.—As the sugar is usually found to be normal in all cases, the tests are ordinarily limited to the fat and protein-contents of the breast milk. After obtaining an ounce of what is called "middle-milk" (*i.e.*, the milk collected after 1 or 2 ounces had been withdrawn) or of the entire breast supply, we determine the following qualifications:—

(*a*) Reaction—fresh breast milk should be alkaline or neutral; may be tested with litmus-paper.

(*b*) Specific gravity—about 1030, taken by means of a lactometer, at a temperature of 65° to 72° F.

(*c*) Fat content—the cylinder of Holt's milk set (see Fig. 24) is filled with the sample of breast milk up to the zero mark and allowed to stand for twenty-four hours in a room temperature of 70°. The percentage of cream is then read off, bearing in mind that the ratio of the cream to the fat is approximately 5 to 3, *i.e.*, 5 per cent. of cream equals 3 per cent. of fat.

(*d*) Proteids—the amount of proteids is approximately determined by the amount of fat and the specific gravity of the milk, *i.e.*, high specific gravity, high proteids; low specific gravity, high fat. Holt's accompanying table explains the application of this principle:—

	SPECIFIC GRAVITY	CREAM—24 HOURS	PROTEID (CALCULATED)
Average	1031	7	1.5%
Normal variations	1028-1029	8%-12%	Normal (rich milk)
Normal variations	1032	5%-6%	Normal (fair milk)
Abnormal variations	Low (below 1028)	High (above 10%)	Normal or slightly below
" "	Low (below 1028)	Low (below 5%)	Very low (very poor milk)
" "	High (above 1032)	High	Very high (very rich milk)
" "	High (above 1032)	Low	Normal (or nearly so)

Reduction of fat in breast milk.

While, as a rule, the breast milk of the modern mother is characteristic for its paucity, we occasionally come across breast milk that is too rich in quality, especially as regards the fat content. In the majority of such instances, if the excess in fat is detected early, it can readily be corrected (by reducing the mother's diet, encouragement of active exercise, etc., or by resorting to partial nursing) before any appreciable harm has been done to the infant. In some cases, however, the abnormality of the milk is not discovered until the infant is suffering from "fat indigestion" (diarrhea with masses of fat, eructations, colic and possibly loss of weight), and one is often in a quandary as to what is best to do. An attempt may be made to thin the breast milk by administering to the infant before each nursing ½ or 1 ounce of plain or cereal water. If this procedure and the dieting of the mother fail, and the child is progressively getting worse, we must either engage a wet-nurse or put the baby on a suitable artificial food.

Diluent before nursing.

Where the milk supply is deficient, partial nursing should be insisted upon, preferably alternating one breast- with one bottle-feeding.

WET-NURSING.

Wet-nurses at best are an evil, but often indispensable, where mothers will not, can not, or must not nurse their own offspring. If the mother cannot nurse her baby because of quantitative or qualitative insufficiency of her milk, there is no urgency of securing a wet-nurse, as the milk may be improved by a richer diet and better care of the mother, or the infant may receive daily two or three feedings of properly modified cows' milk. In the event, however, that the mother is utterly unable to nurse her baby or is prevented from doing it through disease (tuberculosis, cancer; acute, greatly debilitating affections; advanced kidney or heart disease; local inflammation of the breast, psychoses and the like) or pregnancy, a wet-nurse is the best substitute. The wet-nurse to be chosen must undergo a very careful physical examination, first as to the secreting quality of the breasts and the condition of the nipples, and next as to her general health.

Contra-indications to maternal nursing.

The secreting quality of the breast is best tested by grasping it with the thumb and four fingers and, while moving the whole hand somewhat forward, exerting uniform but gentle pressure. With this manipulation the milk should escape from the breast in several even jets for from fifteen to thirty seconds. Too much reliance should not be placed upon the form of the breast, for even pendulous, cylindrical, or conical breasts are occasionally poor milk producers. On the other hand, an abundance of glandular parenchyma offers more reliable guarantee as to its secreting power. The physician should be on his guard that the abundance of milk is not the result of the breast having been allowed to fill up for several hours previous to the examination—a fact recognized by the presence of pain on pressure and intense distention of the

Freely secreting breasts.

Condition of nipples.

mammary ducts. The nipples should be hard, long and bulky, free from *severe* erosions or fissures.

The quality of the milk is not near as essential as the quantity, since the former can usually be improved upon by a suitable diet and good hygiene.

Contra-indications to wet-nursing.

The following diseases render a wet-nurse useless: Tuberculosis, whether local or general; syphilis, in all its stages (not necessarily the mother); non-compensating heart disease, grave affections of all other bodily organs; profound anemia; intractable, communicable skin, hair, eye diseases; gonorrhea; suppurative processes of the bones; mastitis (not necessarily the mother); ozena, drug-addiction, psychoses, and epilepsy.

Guard against syphilis.

The possible presence of syphilis should receive especial attention. Corona veneris, bony tumefactions, nasopharyngeal patches, old ulcers and scars, enlarged glands (especially paramammary, epitrochlear, and inguinal) should invariably arouse the suspicion of the examiner.

The wet-nurse of choice should be one between 20 and 30 years old, who has given birth to two healthy children and nursed one successfully, the age of the last child being nearly the same as the one she is about to nurse. The diet of the wet-nurse, the care of her breasts and nipples, the mode of living, exercise, etc., should be the same as in a nursing mother (*q. v.*).

ARTIFICIAL FEEDING.

Where maternal nursing is impossible, and wet-nursing impracticable, there is nothing else left but to resort to artificial feeding. All human ingenuity and skill have thus far failed to provide a food for infants that is as nutritious, digestible, sterile without interference of composition, and as economic as woman's milk.

Cows' milk best substitute.

With suitable modification cows' milk forms the best substitute for human milk. But it is a poor substitute at best, for not alone does human milk vastly differ from cows' milk in the quantitative proportion of the essential chemical ingredients, but the latter vary greatly also qualitatively. Furthermore, human milk contains several as yet not fully determined biological constituents, especially enzymes, which are absent in cows' milk.

COWS' MILK FEEDING.

To meet the aforementioned requirements of an infant food, cows' milk must undergo elaborate modification, so that its composition conforms, quantitatively and qualitatively, as closely as possible to that of human milk.[1] To accomplish this object, it is, of course, essential to have a clear appreciation of the most important differential peculiarities between the two kinds of milk involved.

Differences between cows' and human milk.

1. Human milk contains approximately half as much proteids as cows' milk.

(*a*) The proteids of human milk are composed of 61.5 per cent. of casein and 38.5 per cent. of albumin against 85.7 per cent. of casein and 14.3 per cent. of albumin in cows' milk. That is, in a given quantity of milk, out of the *total proteid* content, human milk contains approximately 58 parts of albumin and 92 parts of casein, while cows' milk contains 42 parts of albumin and 258 parts of casein.

(*b*) The casein of human milk in the stomach forms loose, flocculent curds, which dissolve readily. On the other hand, the casein of cows' milk coagulates in large, firm clots which are dissolved slowly.

2. Human milk contains 1 to 2 per cent. more sugar than cows' milk.

3. Human milk is sterile, cows' milk replete with bacteria.

4. Human milk is alkaline in reaction, cows' milk neutral or slightly acid.

In addition to these differences human milk contains less volatile fatty acids than cows' milk, and its salt content, while considerably smaller than that of cows' milk, is comparatively richer in iron. With these observations in view, the principles involved in the modification of cows' milk as a suitable infant food are quite obvious. We have to—

Reduce proteids.

1. Reduce the proteids, and more especially render the casein more easily digestible.

This can in part be accomplished by dilution.[2] There

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[1] **Approximate Composition of Human and Cows' Milk.**

	Proteids.	Fat.	Sugar.	Salts.	Water.
Human Milk	1.50	4.00	6.00	0.20	88.80
Cows' Milk	3.00	4.00	5.00	0.70	88.30

[2] **Diluents.**—These are employed with the objects in view: firstly, to thin the milk and thus to reduce the percentage of proteids; secondly, to break up the curd of the casein of cows' milk formed in the infantile

being twice as much of proteid in cows' milk as in human milk, we can add one part of a diluent to every one part of the cows' milk to equalize the proteid value. By doing so we obtain a mixture containing approximately

Proteids	1.50 per cent.
Sugar	2.50 per cent.
Fat	2.00 per cent.

which, while fairly correct in its content of proteid, is greatly deficient in sugar and fat. Our next effort, therefore, must be to—

2. Increase the sugar as well as the fat—both of which having undergone reduction by dilution. Add sugar.

The sugar can readily be replaced by the addition of either cane-sugar (enough to sweeten—about 15 grains) or milk-sugar (about a third of a teaspoonful, or 22 grains, to every ounce of the diluent). The fat may be increased either by adding cream, or, preferably, using top-milk[3] as a base, *i.e.*,

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stomach. Plain water serves the first purpose, *i.e.*, reduces the proteid content of the milk, but has no appreciable effect upon the curd. On the other hand, cereal gruels (carbohydrates) of barley, oatmeal, arrowroot, rice, farina, wheat flour and the different carbohydrate infant foods or "milk modifiers" on the market, answer both requisites. As the capacity for starch digestion in infants under three months of age is very poor, it is not advisable to overburden the young infantile stomach with these products. Small quantities of cereal gruels, however, may be employed with safety and benefit. In infants over six months the diluent may be made up wholly of either barley or oatmeal water—the former especially in the presence of diarrhea, the latter in constipation.

Mode of Preparation of Diluents for Cows' Milk.—*Barley-water.*—One tablespoonful of prepared barley (Robinson's) is rubbed up in a little cold water; to this is gradually added a pint of boiling hot water, and the mixture is allowed to boil slowly (simmer), with constant stirring, for about twenty minutes and then strained. Boiled water is then added sufficient to make one pint.

Oatmeal-water.—One tablespoonful of oatmeal is rubbed up in a little cold water; to this is added a pint of boiling hot water and allowed to boil slowly (simmer) for one to two hours, with frequent stirring, and strained through gauze. Boiled water is then added sufficient to make one pint.

Rice-water.—One tablespoonful of ground rice to a pint of water, prepared the same as oatmeal water.

[3] **Top-milk.**—Bottle-milk, as obtained from reliable milk dealers, contains approximately the following percentages of fat and proteids:—

PORTION TAKEN.	FAT.	PROTEIDS.
Upper ½ ounce.	24.8	3.1
" 1 "	23.1	3.2
" 2 "	21.4	3.3
" 4 "	20.1	3.4
" 6 "	18.6	3.5
" 8 "	16.7	3.6
" 12 "	12.1	3.7
" 16 "	8.4	3.8
" 18 "	6.5	3.9

by taking instead of "whole" milk a sufficient quantity of milk out of the upper 18 ounces of a bottle (which contains about 6 per cent. of fat) decanted for this purpose and thoroughly shaken.

Now, then, by taking one ounce of milk, one ounce of a diluent and a third of a teaspoonful of milk sugar, we obtain a mixture of milk approximately of the following composition:

Proteids	1.50 per cent.
Sugar	6.00 per cent.
Fat	2.00 per cent.

Which, though still deficient in fat (the fad of high percentages of fat has, as a result of frequent occurrence of "fat indigestion" in infants, lost its strongest advocates), is nevertheless amply nutritious and well tolerated by infants.

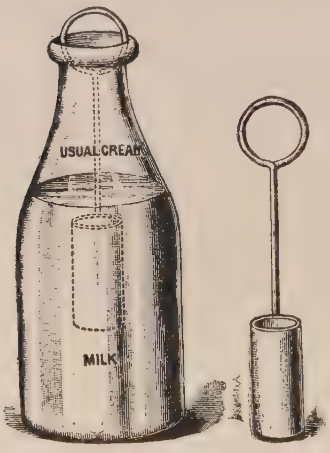

Fig. 25.—Chapin's Dipper for Removal of "Top-milk."

3. Render the milk alkaline. For this purpose we resort to either ½ grain of sodium bicarb. to every ounce of milk or to 5 per cent. of lime-water. Both of these preparations have the additional qualification of preventing the casein from coagulating into tough curds.[1]

4. Obtain and maintain milk at a certain standard of cleanliness and insure the absence of pathogenic bacteria.

It is well to bear in mind that the milk of a healthy cow is as free from bacteria while still in the udder as that of a woman in the breast. Contamination of cows' milk occurs from local affections of the udder or the teats, dirty utensils, the dust of the stable, the hands of the milker or others engaged at the dairy, from flies or insects, droppings of particles of manure from the tail or belly of the cow, from polluted water employed in adulterating the milk, etc. The rendering of milk free from bacteria, therefore, must begin long before the milk has been distributed to the consumer.

Contamination of cows' milk.

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[1] Sodium citrate (gr. ij to every ounce of milk) answers the same purpose.

The cow must be free from disease, especially from tuberculosis as determined by the tuberculin test and by regular inspection by a competent veterinary surgeon.

Cleanliness of the stable, cow, and milker.

The cow's entire body should be groomed daily, and immediately before milking the belly, tail, and particularly the udder should be carefully cleansed with a clean, damp cloth, with or without soap, and dried with a clean towel.

The milker must be free from communicable affections. Before milking he should thoroughly scrub and dry his hands and don clean, washable, outer garments. He should have a few of these on hand, in order to change them should one gown or suit accidentally get soiled in the act of milking.

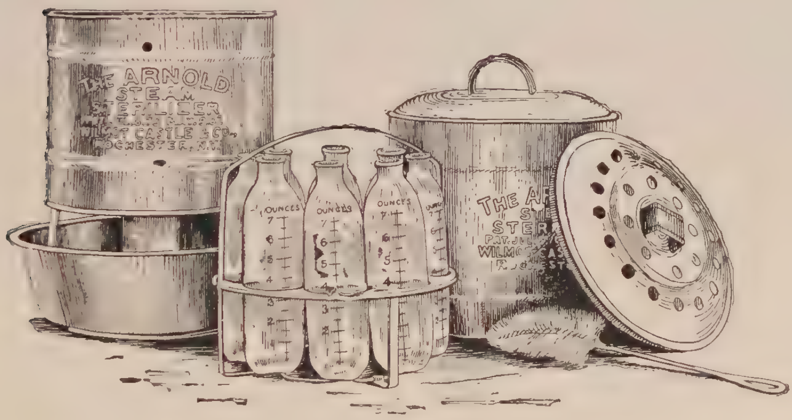

Fig. 26.—Arnold Steam Sterilizer.

The milk of each cow should be collected separately in sterile utensils and immediately removed from the stable to a specially clean place reserved for the keeping of the milk until ready for shipment.

Cooling of milk.

The milk should be rapidly cooled (below 45° F.) and strained through a sterile strainer, then bottled, closed with sterile discs, capped, and finally iced—all within an hour or so after milking.

After reaching the consumer, which, owing to the rapid development of bacteria in milk over twenty-four hours old, should occur within this period of time, the milk is further kept on ice until needed for the preparation of the food.

If, notwithstanding all the prophylactic measures, some doubt still remains as to the sterility of the milk, we must subject it to sterilization or pasteurization.[1]

LABORATORY AND HOME MODIFICATION OF COWS' MILK.

We have just learned the numerous essential differences of composition that exist between human and cows' milk, and the means by which the differences can be removed. Were it merely a question of obtaining milk of a definite uniform, composition which would at once prove suitable for the feeding of infants of all ages, the problem of artificial feeding of infants would long have been solved. Unfortunately this is not the case. Not only must cows' milk be modified so that its principal constituents as closely as possible resemble those of human milk, but it must undergo also specific modification to meet the digestive powers and the requirements of the individual infant at certain periods of life—quite a difficult proposition indeed, yet within the scope of execution.

Modification of milk to meet the needs of the individual baby.

Thanks to the rapid strides of physiological chemistry, and the good will and enterprise of several milk dealers and laboratory chemists, the modification of cows' milk as an infant food has almost reached a stage of perfection. With the help of the laboratory chemist, the physician is now enabled to write a prescription for a food mixture of definite composition and, like a drug in the pharmacy, have it compounded exactly as ordered. The latitude of composition is well illustrated in the prescription here appended:—

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[1] **Sterilization and Pasteurization.**—Both of these processes are accomplished by means of one of the many sterilizers on the market. In *sterilizing* the milk is heated for about fifteen minutes at a temperature of 212° F.; in *pasteurizing,* for about forty minutes at a temperature of from 140° to 150° F. For infant-feeding the milk should undergo the heating process after it has been modified and divided in the requisite number of feeding bottle for the entire 24 hours. The bottles are cooled off by allowing cold water slowly to run through the sterilizer; they are then tightly corked, preferably with non-absorbent cotton, and placed on ice until needed for use. Before feeding the bottle should be warmed to body heat. Except during the hot summer months or when there is good reason to believe that the milk harbors virulent bacteria (*e.g.,* during epidemics of typhoid, cholera, etc.), sterilization is nowadays rarely practised. Pasteurization is usually resorted to instead, particularly since it has been demonstrated that this process is less apt to change the taste of the milk, to interfere with its digestibility, and to cause constipation. The view held, especially by overenthusiastic, though well-meaning, laymen, that pasteurized milk is as nutritious as clean, fresh, raw cows' milk, is not based upon scientific observation. Quite the contrary; pasteurized milk lacks several nutritive and protective elements that exist in fresh cows' milk. Hence, its continued use greatly interferes with the growth and development of the infant, and is not rarely productive of rickets and scurvy.

℞	Per cent.			
			Number of feedings	7
Fats	2	......		
Carbohydrates—lactose (milk-sugar)	6	......	Amount at each feeding	4½ ℥
Dextrinize	......	......		
Proteids } Whey	......	75	Other diluent—Barley-water	50 %
Proteids } Casein	......	75		
Peptonize	......	......		
Lime-water % of total mixture	5	......		
Sodium citrate } % of milk and cream	......	......	Heat at	°F
Sodium citrate } % of total mixture	......	......	Raw	
Sodium bicarb. } % of milk and cream	......	......		
Sodium bicarb. } % of total mixture	......	......		
Lactic acid bacillus				

Home modification of cows' milk, after standard formula.

When "laboratory milk" is not obtainable and "home modification" has to be resorted to, we can greatly facilitate this process and obviate the difficult task of memorizing complicated formulas by selecting a milk formula of simplest composition (1 : 1, *i.e.,* 1 ounce or its multiple of milk to 1 ounce or its multiple of a diluent, in which are included one teaspoonful of lime-water for every ounce of *milk*, and one-third of a teaspoonful of milk-sugar for every ounce of the *diluent*) and preparing the other milk mixtures by modifying this "standard" formula.

Directions.—1. Bear in mind the standard formula (1 : 1), which is intended for babies six months old.

2. For infants *under* six months increase (about every two months downward) the *diluent* by one ounce or its multiple, using "top milk" as a base and plain or cereal water as the diluent.

3. For infants *over* six months of age, increase (every two months upward) the *milk* by one ounce or its multiple, using "whole milk" as the base and cereal water as the diluent.

4. Include in the diluent one teaspoonful of lime-water for every ounce of milk, and add one-third of a teaspoonful of milk-sugar for every ounce of the diluent.

In accord with these directions the following milk formulas are obtained:—

1:2 four months		2:1 eight months
1:3 two months		3:1 ten months
	Standard Formula 1:1 for an infant six months old.	
1:4 one month		4:1 twelve months
1:5 half a month		5:1 fourteen months

Milk modified in accordance with this table yields mixtures *approximately* of the following composition:—

For an infant 2 weeks old—(1:5).

Top milk	1 ounce	Proteids	0.50
Lime-water[1]	1 dram	Sugar	6.00
Diluent (water)	5 ounces	Fat	1.00
Milk-sugar	1⅔ drams		

For an infant 1 month old—(1:4).

Top milk	1 ounce	Proteids	0.60
Lime-water	1 dram	Sugar	6.00
Diluent (water)	4 ounces	Fat	1.20
Milk-sugar	1⅓ drams		

For an infant 2 months old—(1:3).

Top milk	1 ounce	Proteids	0.75
Lime-water	1 dram	Sugar	6.00
Diluent (25% barley water)	3 ounces	Fat	1.50
Milk-sugar	1 dram		

For an infant 4 months old—(1:2).

Top milk	1 ounce	Proteids	1.00
Lime-water	1 dram	Sugar	6.00
Diluent (50% barley water)	2 ounces	Fat	2.00
Milk-sugar	⅔ dram		

For an infant 6 months old—(1:1).[2]

Whole milk	1 ounce	Proteids	1.50
Lime-water	1 dram	Sugar	6.00
Diluent (barley water, 75%)	1 ounce	Fat	2.00
Milk-sugar	⅓ dram		

For an infant 8 months old—(2:1).

Whole milk	2 ounces	Proteids	2.00
Lime-water	2 drams	Sugar	6.00
Diluent (barley water)	1 ounce	Fat	2.33
Milk-sugar	⅓ dram		

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[1] To simplify the formulas the lime-water is not included in the diluent.

[2] Standard formula.

For an infant 10 *months old*—(3:1).

Whole milk	3 ounces	Proteids	2.25
Lime-water	3 drams	Sugar	6.00
Diluent (barley water)	1 ounce	Fat	3.00
Milk-sugar	⅓ dram		

For an infant 12 *months old*—(4:1).

Whole milk	4 ounces	Proteids	2.40
Lime-water	4 drams	Sugar	6.00
Diluent (barley water)	1 ounce	Fat	3.20
Milk-sugar	⅔ dram		

For an infant 14 *months old*—(5:1).

Whole milk	5 ounces	Proteids	2.50
Lime-water	5 drams	Sugar	6.00
Diluent (barley water)	1 dram	Fat	4.00
Milk-sugar	⅔ dram		

Advantages of this method.

It is quite obvious that the method of home modification of cows' milk, just described, is very far from being exact. The same, however, applies also to all the other methods in vogue. The latter, furthermore, have the disadvantage of requiring the knowledge of higher mathematics for their compilation. It will be noted that the milk mixtures, especially those intended for infants under six months of age, are comparatively poor in proteids and fat. From my extraordinarily large experience in infant feeding, as chief of one of the milk depots of the Charity Organization Society, I have learned to know that infants invariably do better on weak mixtures than on stronger ones. With the advance of the infant's age and its demands for a more liberal food supply the latter can readily be furnished by a moderate allowance of other articles of food, such as bread, soup, cereals, eggs, etc., as fully outlined in this chapter (see page 80).

QUANTITY OF FOOD.

Varies with size of baby and capacity of stomach.

The keynote to successful feeding is avoidance of overfeeding. Infants almost always *act* hungry, though in reality *are* so only at certain times. Colicky babies particularly never seem to have enough of food, and the more they drink the greater the colic and *vice versâ*—the old story again of the "vicious circle." The amount of food needed by the healthy infant is best judged by the capacity of its stomach. This is subject to great variation, ranging vastly not alone with the age of the infant, but also with its general development and the state of tonicity of the muscular walls.

FEEDING SCHEME.
(For 24 hours)

Age of infant in months	Formula	Feeding intervals in hours	Number of feedings	Size of feeding in ounces	Ingredients for total number of feedings (in ounces)						Approximate pecentage composition		
					Milk		Lime-water	Diluent		Milk-sugar in teaspoons			
					Top[1]	Whole[2]		Water	Cereal		Proteids	Sugar	Fat
½	1:5	2½	8	2	2⅔	..	⅓	13	..	4⅓	0.50	6	1.00
1	1:4	2½	8	2½	4	..	½	15½	..	5⅓	0.60	6	1.20
2	1:3	3	7	3½	6¼	..	¾	13½	4	6	0.75	6	1.50
4	1:2	3	7	4½	10½	..	1¼	9¾	10	7	1.00	6	2.00
6	1:1	3	7	6	..	21	2¾	..	18⅓	7	1.50	6	2.00
8	2:1	3½	6	7	..	28	3½	..	10½	4⅔	2.00	6	2.33
10	3:1	3½	6	7½	..	33¾	4¼	..	7	3⅔	2.25	6	3.00
12	4:1	4	5	8½	..	34	4½	..	4½	3	2.40	6	3.20
14	5:1	4	5	9	..	37½	4¾	..	3	2⅔	2 50	6	4.00

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[1] The upper eighteen ounces of a quart bottle = 6 per cent. fat, 5 per cent. sugar, 3 per cent. proteids.

[2] 4 per cent. fat; 5 per cent. sugar; 3 per cent. proteids.

Stomach capacity.

The child should receive with each feeding as much food as the stomach will hold[1] minus half to two ounces to avoid its overflowing. It is practicable to prepare, every morning, the total amount of food for twenty-four hours; to divide it in the requisite number of feedings in separate bottles, corked with non-absorbent cotton, and—preceded or not by sterilization or pasteurization—to place it and keep it on ice until needed for feeding.

As a rule, properly modified milk in suitable quantities agrees well with the great majority of infants, especially if its administration is started long before their digestive tracts had been ruined by the domestic feeding-panaceas—which, as usually, served to "make bouncers" of some neighboring children or "raised a family of twelve." However, occasionally infants do show a certain idiosyncrasy to cows' milk, regardless of its quality or quantity, and if a wet-nurse is not procurable, we have to resort to other food preparations to sustain the infants' lives—perhaps long enough until modified milk is tolerated.

COWS' MILK SUBSTITUTES.

Different milk substitutes and milk modifiers have from time to time been recommended in "difficult feeding cases." We will enumerate them in the order of their usefulness.)

Malt-soup.—Two ounces of wheat flour are slowly and thoroughly mixed with one pint of milk, and strained through gauze. In a second vessel three ounces of thick malt are dissolved in a pint of warm water to which had been added fifteen grains of carbonate of potassium. Now both solutions are mixed together and heated very slowly up to a boil. For delicate infants this preparation may be diluted by an equal or smaller quantity of water. Malt-soup is often particularly beneficial in underfed, dyspeptic and rachitic babies. If well tolerated it may be continued for several months.

Dyspepsia and marasmus.

As the infant improves and can tolerate a heavier food, the quantity of milk may gradually be increased—*without* a corresponding addition of flour or malt.

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[1] The following fairly represents the *average capacity of the infantile stomach*: At the end of the first week, 1 ounce; the second week, 2 ounces; first month, 3 ounces; second month, 4 ounces; fourth month, 5 ounces; sixth month, 6 ounces; eighth month, 7 ounces; tenth month, 8 ounces; twelfth month, 9 ounces; fourteenth month, 10 ounces.

Condensed Milk.—Where the principal difficulty consists in incapacity to digest cows' milk casein, condensed milk[1] will be found to act kindly, since the consistency of the coagulum of condensed-milk casein formed in the infantile stomach greatly resembles that of human milk. It has also the advantages of being inexpensive and not as readily subject to contamination as ordinary cows' milk. However, containing as it does about 51 per cent. of sugar, and requiring eight to ten times dilution to approximate the sugar content of human milk, the simultaneous reduction (by dilution) of the fat and proteid contents to about 1 per cent. and 1¼ per cent. respectively renders condensed milk too poor in quality to serve as an ideal infant food. Indeed, it is usually found that infants over three months fed on diluted condensed milk soon contract rachitis. Nevertheless, as a temporary food, especially during the summer months or a long journey, it is invaluable. As already suggested condensed milk should be administered in quantities appropriate for the infant's age, in dilution with from eight to ten or even twelve parts of plain or cereal water. The deficiency of fat may be supplemented by the addition of cream.

As a temporary food.

Whey.—Where the digestive capacity of casein is greatly at fault, we may temporarily resort to whey feeding. Whey is obtained by adding to a pint of fresh warm (100° F.) milk two teaspoonfuls of essence of pepsin. After it stiffens beat up the curd with a fork and strain through a few layers of gauze so as to withhold the coagulated casein. The decanted liquid contains approximately:—

	Proteids.	Sugar.	Fat.
Lactalbumin	0.9%	4.5%	0.5%
Casein	0.3%		

By adding a little sugar to overcome its deficiency and employing a cereal diluent instead of plain water, the whey mixture is amply nutritious to sustain an infant's vitality for several weeks or months. Whey is most useful in digestive disturbances requiring low proteids, in mixed breast- and bottle-feeding, in summer diarrheas, and in acute fevers. In fact, one of the principal advantages of "laboratory milk" is the adoption of whey as its chief proteid component.

In diarrhea and proteid indigestion.

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[1] Approximate composition of condensed milk:—

Proteids.	Sugar.	Fat.	Salts.	Water.
8.00	51.00	7	1.50	32.00

Buttermilk.—This is prepared by thoroughly mixing, in a suitable agate vessel, one quart of fresh, rich milk, with a pint or less of water, a pinch of salt, and the pure lactic acid culture (any of the pure mercantile lactic bacilli tablets answers the purpose). The vessel is covered with a thin cloth and allowed to stand in the room (70° to 80° F.) for from eighteen to twenty-four hours. It is now placed on ice until needed. For infant feeding we add to every quart of buttermilk a flat tablespoonful of wheat-flour and two tablespoonfuls of cane-sugar and allow the mixture to *boil* over a low fire, for two to three minutes, with constant stirring. The food is now poured, in quantities varying with the age of the patient, in sterilized bottles, properly corked, and placed on ice until used. This mixture is indicated especially in cases requiring a high percentage of protein and a low percentage of fat, *e.g.*, gastroenteritis and fat indigestion.

In fat indigestion.

Proprietary Milk Modifiers and Milk Foods.—We distinguish two kinds of proprietary foods—milk modifiers, and so-called milk foods. Neither of them contains a sufficient amount of nutrient elements to supply the needs of the baby for life and growth for any length of time; they are useful, however, in digestive disturbances and "milk idiosyncrasy," and to bridge over an acute siege of sickness. The mercantile milk modifiers furnish soluble carbohydrates, free starch, or predigested proteids in small quantities, and thus save the trouble of home-preparation of suitable diluents. Contrary to what is generally expected, the so-called milk foods *occasionally* prove very beneficial in cases of pedatrophy. On the other hand, it should be remembered that their prolonged use is frequently followed by scurvy and rickets.

In milk idiosyncrasy.

Peptonized Milk.—The use of peptonized milk is nowadays limited chiefly to feeding of children of very low vitality, in whom the powers of digestion are in abeyance, *e.g.*, high fever, coma (administered in the form of nutrient enemata, or by gavage), pyloric stenosis, etc.

In very poor digestion.

Mode of Preparation.—Mix in a quart bottle one pint of fresh milk with four ounces of cold water containing 5 grains of pancreatic extract and 15 grains of sodium bicarbonate, or the contents of one of Fairchild's peptonizing tubes. Place the bottle in a pot of hot water and maintain its temperature at about 115° F., either for about twenty minutes ("partial" peptonization) or two hours ("complete" peptonization). Shake the bottle from time to time. When the mixture is ready, give it, either pure or diluted, in quantities suitable for the age of the child. Keep it on ice until used.

WEANING OF THE BABY AND ITS FEEDING THEREAFTER.

Time for weaning.

Ordinarily it is not advisable to nurse an infant beyond ten or eleven months old. As exceptions to this rule, we may mention the very hot summer months, acute diseases, difficult teething, etc., when a complete change in feeding is prone to prove hazardous to the child's health. It is preferable to wean a baby gradually, by substituting bottle- for breast-feedings, and to continue to partially nurse it, until the infant has learned to submit to the inevitable, and thrives well on the new food.

Gradual transition from milk to other articles of food.

Feeding of Infants Over Ten Months Old.—When the normal infant reaches the age of ten months or thereabouts, nature announces the urgency of a change in the dietary—from liquid to solid—by hastening the eruption of the lower and upper incisors which for months had threatened to escape from their seat of birth and captivity. At this age also salivary digestion is fully established, so that an allowance, once or twice a day, of a crust of stale or toasted bread, or zwieback, certainly can do no harm. As at this period of life the tendency to rickets is very pronounced, the dietary should be gradually improved upon by the addition of cereals, a teaspoonful or more of fresh soft-boiled egg, oatmeal or graham crackers; strained chicken, mutton or beef soup, with fresh vegetables (*e.g.,* carrots, potatoes, etc.), orange- or pineapple-juice, and later baked apple, baked potato with some sweet cream or butter, bread and butter, milk custards, cocoa and occasionally finely scraped beef or chicken.

Of course, the transition from an exclusive milk diet to a more or less mixed diet must be very slow and gradual. The effect of the change should be watched from day to day and week to week, always bearing in mind that milk is the ideal food for the infant and indispensable to the child up to the period of second dentition.

This fact should be strongly impressed upon those in charge of the child, as only too often, with the allowance of a semisolid diet milk is crowded out entirely by an oversupply of thin soups, indigestible, proprietary "breakfast foods," and all sorts of sweets and fruit of poor quality, which sooner or later upset the child's digestive powers and arrest its growth and development—doing just the opposite of what the change of diet was intended for.

Milk to remain the principal foodstuff.

With the change in the diet also it is frequently observed that the infants refuse to drink milk. Inquiry into the cause usually reveals the fact that upon the advice of some artistically inclined neighbor—who thinks that the bottle effaces the child's "beauty lines"—and more generally upon the recommendation of the family physician, the child is forced to part with its bottle and nipple—its dear and faithful companions for the many months past. Why milk-bottles are to be looked upon as an abomination for children *over* a year or so and as a salvation for those *under* this age, is to me a mystery. The mere facts that if given in a bottle, large quantities of milk are enjoyed by children up to four or five years of age; that if taken through a nipple, milk enters the stomach slowly, and, hence, is more easily digested, and, finally, that during sickness milk (as well as water) is best administered through a bottle, are ample justifications for the encouragement rather than the prohibition of the use of the bottle—provided, of course, that the bottles, as well as the nipples, are kept scrupulously clean; are sterilized, if you please.

Advantages of giving milk through a bottle.

The additional articles of food should be given at definite intervals, preferably together with the milk feeding. Thus, for example, with the ten o'clock bottle the child should receive the soft-boiled or poached egg and crackers; at two o'clock the meat broth and potato; at six o'clock, some cereal and bread and butter. Orange- or pineapple-juice may also be given between meals. The child should be taught to appreciate that to get other food-stuffs it must drink its allowance of milk.

Feeding of Children of from Two to Four Years Old.—With completion of primary dentition, which usually occurs between the twenty-fourth and thirtieth months, the dietary of the child can be considerably enlarged. The breakfast in addition to the milk should consist of well-cooked cereal gruel, toasted bread and butter, scraped or baked apple. The dinner may embrace meat broths, broiled rare steak, mutton chop or white meat of chicken—all cut up finely; scraped raw beef,

Breakfast.

Dinner.

boiled fish; small quantities of vegetables, such as potato, fresh string beans or peas, spinach or asparagus tops, stewed fruit or a little custard or pudding. The supper should include a soft-boiled or poached egg, bread and butter, stewed fruit, and milk or cocoa. The child should receive a cup of milk between breakfast and dinner and between dinner and supper. Fresh, pure water should be given between meals, the first thing in the morning and the last thing at night.

Supper.

Feeding of Children from Four to Six Years Old.—The dietary of children over four years old is practically identical with that just mentioned, except that the quantity of the food should be more liberal, the fruit may be given raw, and that the between-meals milk allowance should be dispensed with. Occasionally the child may receive home-made cake, a little ice cream and other condiments of good quality. All these food-stuffs, however, should be given with regular meals.

More liberal diet.

II. HYGIENE AND SANITATION.

Next to suitable nutrition hygiene and sanitation play the most important rôle in the preservation of good health. It is within the province of the physician's duties to formulate, to those intrusted with the care of the child, rules and regulations as to its cleanliness and comfort, mode of clothing, time for sleeping, airing, bathing, rest and exercise, both during health and disease. Without the advice and supervision of the physician, the nurse or mother is only too apt to either overdo or underdo, *i.e.*, in both events do irreparable damage to the health and welfare of the child. The period of blind credulity, stupid mysticism and absurd fatalism still reigns supreme, the great strides in science and adventure notwithstanding.

GENERAL CARE OF THE NEWLY BORN AND OLDER CHILDREN.

The Newly Born Baby.—Immediately after birth the infant instinctively, by its shrill cry, announces its demand for protection against the sharp change of atmosphere and surroundings. Therefore, after dressing its navel (see page 173), washing its eyes and mouth with a saturated boric acid solution,[1] the baby should be wrapped in a warm woolen blanket and placed

Attention to navel and eyes.

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[1] Where gonorrhea in the mother is suspected, we should instill into each eye one drop of a 2 per cent. solution of nitrate of silver.

Rest. **Nursing.**

in a warm, darkened, but airy, quiet room, and left to rest for a few hours. It should then be sponged off with warm soap water, dressed, given a little clean water, and, the condition of the mother permitting, put to the breast (see page 62). Wherever possible, the child's crib should be kept in a room apart from that of the mother, so that the latter is not disturbed by the possible uneasiness experienced by the baby. As lactation is usually not fully established before the third or fourth day after labor, the infant should, in the mean time, several times daily receive a few teaspoonfuls of plain or slightly sweetened warm water or of a mild carminative, such as fennel-seed tea, to satisfy its thirst and hunger.

Tea of aromatic
peppermint
spearmint
[illegible]

22 mt 82

Hours of sleep.

Sleep.—The normal newly born baby sleeps practically all the time except the brief periods occupied with nursing, diapering, and dressing. If the baby is well developed and strong, it should be left to sleep until it wakes up of its own accord from hunger; if delicate it should be aroused every two hours during the day, and once at night, made to cry a little to help expanding its lungs and put to the breast for from ten to twenty minutes. At six weeks the infant needs twenty hours of sleep; at three months eighteen; at one year sixteen and from two to four years fourteen hours of sleep. All children should get accustomed to sleep uninterruptedly (except for one nursing in the middle of the night in early infancy), from seven in the evening until seven o'clock in the morning, and one hour each sometime between seven and twelve o'clock in the forenoon and two and seven in the afternoon.

Sleeplessness in the infant is ordinarily due to intestinal colic or other pain, discomfort from soiled diapers or faulty dressing (overheating by superabundance of clothes, etc.), noise in the room, lack of ventilation, bad habits, such as rocking, or keeping an empty nipple in the mouth, etc. Repeated waking is frequently due to over- or under-feeding.

Bathing to be postponed.

Bathing.—In view of possible local or systemic infection (see page 172) through the umbilical rest, and the advisability of keeping the latter perfectly dry, the full tub-bath should be withheld until the navel has completely healed. The same applies for circumcision wounds. In the mean time the infant should receive at least one sponge bath a day, to be given as gently as possible, since the infantile skin is very delicate, very apt to be abraded on rough handling, and readily becomes subject to divers skin affections.

Clean bath-water.

In the absence of the aforementioned or other contraindications, every child, in addition to local cleansing as frequently as necessity arises, should receive a tub-bath once a day, preferably at bedtime. The water used should be free from visible impurities, and obtained from sources inaccessible to pollution. The temperature of the water should range between 95° F. and 98° F., the latter for infants under six months, and cooler water for older ones. Fat babies tolerate much lower temperatures, but I see no special benefit to be derived from the use of bath-water under 95° F. unless it be in the open sea or ocean (which is permissible in children over three years of age), where the saline ingredients and forceful current exert a stimulating, refreshing effect upon the system and thus counteract the depression produced by the sudden lowering of the body temperature. If cool bathing is desirable it is better to place the child in warm water and either to gradually cool off the water while the child is in the tub or use a cold shower. The bath should be followed by thorough drying of the body and gentle friction. Care should be exercised in the selection of pure, non-irritating bathing soap, lest its irritating ingredients may prove a source of annoying skin eruptions. For the same reason and, furthermore, owing to the fact that they are apt to harbor dirt and disease, the use of sponges is to be deprecated.

Temperature of the water.

Avoidance of irritation of the skin.

Clothing.—Infants should be clothed warmly and simply, free from fancy frocks and frills, strings and bows, that embarrass free motion, breathing, sleeping and eating. The underwear should be made of silk or thin flannel. The abdomen should be protected against being chilled by a flannel band. The consistency of the outer clothing should vary with the changes of the weather and season of the year. The feet of infants should at all times be kept warm, if necessary, by means of a hot-water bag. The night clothes should be loose and warm, and consist, in addition to a small silk or flannel shirt, Canton flannel or stockinet diaper and the belly-band, of a nightshirt in the form of a "bag" that buttons around the neck and can be closed at the feet by means of drawstrings. In this manner the unnecessary piling up of blankets, to keep the baby from uncovering, can advantageously be dispensed with.

Silk or flannel underwear.

Night bag.

Older children should gradually get accustomed to light clothes—linen or silk undergarment, linen or woolen suit or dress, and for the winter a warm top coat and cap—but no collars or neck mufflers. A woolen union suit with feet for the night. Especial attention should be paid to the selection of shoes. They should comfortably fit the feet and allow spreading of the toes. The stockings should be fastened to the drawers, as garters are apt to interfere with the blood circulation of the lower extremities. The corset should be prohibited in girls under fourteen.

Shoes to fit the feet.

Airing.—Fresh, pure air is the panacea for good health, the cure of all bodily ills. Thus far it is non-assessable, non-taxable, and hence should be inhaled *ad libitum*—while this freedom lasts. Weather permitting it should be inhaled out of doors, otherwise indoors—in properly ventilated rooms. The newly born baby should be taken out of doors in the summer when it is two weeks old, in the spring and fall at one month and in the winter at two months of age or later. It should be suitably dressed and protected from undue exposure to the sun and wind and severe cold. It is foolhardy to expose an infant to marked atmospheric changes without proper shelter, merely for the purpose of "hardening" it. Its first airing should last from fifteen to thirty minutes, and as it grows older the airing time should be lengthened so that, weather permitting, the child may live out of doors the greater part of the day from sunrise until sunset. Slight rain or snow forms no hindrance to taking the baby out of doors, although in such weather delicate babies do better if aired indoors, in front of open windows and dressed as for outdoors.

Fresh air at all times; outdoor air only when weather permits.

Exercise.—A healthy infant, if not immobilized by burdensome clothes, begins to take physical exercise soon after birth. It kicks, moves its arms and head and exercises its thoracic muscles while crying lustily, especially when feeding time approaches. It should be picked up in the arms at every nursing to insure change of position. At about four months of age the baby is able to hold its head erect; it may then be gradually trained to sit upright upon the arm of the nurse with the hand of the other side supporting its back and head. As it reaches the age of seven or eight months, the infant may be seated alone in a baby-chair supported with pillows at the back and sides. When it shows an effort to creep, it may be

Frequent change of position.

Holding head erect.

Sitting and creeping.

placed upon the floor, which should be well covered by thick carpet or a blanket, preferably within a small portable "creeping pen," and allowed to roam about for half an hour at a time once or twice a day. Less freedom should be granted an infant in its first attempts to stand or walk. These practices should not be encouraged in babies under one year of age, nor in older children who show a tendency to bony curvatures and rickets. In the beginning they should not be allowed to stand or walk, especially if unsupported, for more than a few minutes at a time. But, as they grow older and stronger they are gradually permitted to enjoy shorter or longer outdoor walks and to romp merrily, giving vent to that characteristic boundless joyousness of early childhood which is blessedly ignorant of the pangs and pains of later life.

Standing and walking.

Older children, like infants, should spend the greater portion of the day outdoors in parks and play-grounds and engage in amusing games and light calisthenics which will keep them from harm and mischief. It is opportune on this occasion to emphasize the danger of overindulgence in the practice of gymnastics, especially in children of school age—a period of life which is coincident with prevalence of communicable diseases and their grave sequelæ, particularly cardiac involvement. Carried away by the enthusiasm over the daring feats of the author and exponent of "strenuous life," the young and old alike have recently rushed for rough athleticism with a vengeance, that is daily reflected by multitudes of crippled, so-called athletic, hearts, and apt to become a menace to the health and welfare of our country.

Danger of over-indulgence in athletics.

It is the duty of the physician to impress upon those under his care that while moderate exercise, especially walking, skating and horse-back riding; the daily use, for about fifteen minutes at a time, of light wooden dumb-bells, light clubs or wands; the practice of breathing (see page 353), of swinging of the body from a swinging bar or rings and straps, will do much for the development of delicate and narrow chests and to prevent and straighten curvatures of the spine, stooping of the shoulders, and the like (and should be encouraged), violent sports, like racing, rough baseball- and football-playing, leaping, prolonged swimming and similar severe exercises indulged in to excess, will sooner or later lead to cardiac hypertrophy with its consequences.

Value of moderate exercise.

Air space. **Temperature.** **Ventilation.**

Nursery.—As infants and older children spend about two-thirds or more of their time of life in the nursery, provisions must be made that the room is spacious and airy, dry and sunny, that its air is fresh and pure, free from obnoxious odors, gases, dust and smoke. To thrive well an infant requires about 1000 cubic feet of air space. The room should not be crowded with dust gatherers, *i.e.*, overabundance of furniture, toys, heavy hangings, carpets, rugs, pictures, etc. The temperature of the room should be about 70° F. during the day and about 65° F. during the night. Whenever possible it should be heated from an open fire-place or a hot-air furnace. Steam heat or gas greatly vitiates the air. To insure proper ventilation, it is advisable to keep the windows more or less open from top and bottom most of the time unless the outdoor temperature is below 35° F. The windows and doors should be widely opened while the child is out of doors, otherwise ventilation should be accomplished with the doors closed to avoid draughts. For the latter purpose one of the many ventilating devices on the market will prove very serviceable.

Financial circumstances permitting, every child should have a separate room, if possible, situated one floor above the ground. Of course, this is rarely attainable in the dingy apartments of overcrowded cities. Physicians should insist, however, on every child having a separate bed in order to minimize the danger of transmitting communicable diseases from the sick to the healthy child.

Dust gatherers.

The Sick-room.—The hygienic suggestions just made in reference to the nursery apply with greater force to the sick-room. If possible, the latter should be situated on a different floor from the living apartments. From a sanitary as well as economic point of view it is essential to have the sick-room cleared of curtains, tapestries, superfluous furniture, carpets, etc., so as to facilitate keeping the room perfectly clean, and to prevent pathogenic germs (*e.g.*, with the skin-peeling of scarlatina), becoming firmly imbedded in those articles. The floor and furniture of the sick-room should be wiped off with a damp cloth instead of dusted or swept.

Ante-room.

An ante-room is a useful addition to a sick-room, especially when the patient is suffering from a communicable affection, as it enables the nurse to disinfect the dishes, soiled bed-clothes, linen, etc., and to prepare some of the patient's food.

When the isolation-period of the patient is over, the sick-room, ante-room and their contents must undergo very thorough cleaning and disinfection.

Quarantine and Disinfection.—In order to prevent spreading of communicable diseases from one individual to another, we have to resort principally to the following prophylactic measures:—

1. Isolation of the patient.

2. Disinfection of the patient's excretions, fomites, etc., coming in contact with the pathogenic micro-organisms.

3. Exclusion and destruction of other germ-carriers, *e.g.*, mosquitoes, flies and fleas.

1. Isolation of the Patient.—This is the most essential and efficient mode of prevention of transmission of disease. The isolation to be effective must begin early and be complete.

Early and complete isolation.

In hospitals and asylums every child should be isolated in an observation ward for at least three days before being permitted to mingle with the other inmates; in private families isolation should be enforced with the earliest appearance of tangible symptoms of the specific affection. As those coming in close contact with the patient are apt to carry the disease from the sick to the well, it is imperative to isolate the nurse together with the patient and to forbid any member of the family to stay around the sick-room or make herself generally useful unless on entering the sick-room she dons a clean gown and cap, and before leaving it washes her hands and forearms with soap and water and removes the gown and cap. These latter rules should be complied with also by the physician.

In a private dwelling, and especially in houses where a room is reserved for the sick, perfect isolation can readily be insured. In crowded tenement rooms, however, with people in poor circumstances, all attempts at isolation almost invariably fail, and where the spreading of a grave, epidemic affection is concerned (*e.g.*, small-pox, cerebrospinal meningitis), should not at all be attempted. In such cases it is best to remove the patient to a hospital for contagious diseases.

Removal to hospital.

The period of isolation varies, of course, with different diseases and the degree of severity. The following suggestions will meet the ordinary requirements as to the period of isolation and the principal mode of prophylaxis:—

Periods of isolation in different communicable diseases.

In typhoid fever, while the disease lasts. (Disinfection of excreta; protection against flies.)

In typhus fever, while the disease lasts. (Free ventilation.)

In miliary tuberculosis, while the disease lasts. (Disinfection of excreta.)

In epidemic cerebrospinal meningitis, while the disease lasts. (Disinfection of discharges.)

In yellow fever, while the disease lasts. (Destruction of mosquitoes.)

In relapsing fever, while the disease lasts. (Destruction of insects.)

In influenza, pneumonia and pulmonary tuberculosis, while the diseases last. (Disinfection of discharges.)

In bubonic plague, about one week after termination of the disease. (Destruction of vermin, especially rats; disinfection of excreta.)

In cholera Asiatica and epidemic dysentery, one week after termination of the disease. (Disinfection of excreta; avoidance of pollution of water, milk, etc.)

In small-pox, six weeks. (Vaccination, disinfection of discharges.)

In chicken-pox, three weeks. (Disinfection of discharges and skin.)

In measles, two weeks. (Disinfection of discharges and skin.)

In German measles, two weeks. (Disinfection of discharges and skin.)

In diphtheria, as long as diphtheria bacilli abound in the throat. (Disinfection of discharges.)

In scarlet fever, while the desquamation lasts. (Disinfection of discharges and skin.)

In whooping-cough, while whoop or vomiting lasts. (Disinfection of expectoration.)

In mumps, three weeks. (Disinfection of sputum.)

In erysipelas, two weeks. (Disinfection of the skin; antiseptic dressing.)

In gonorrheal ophthalmia or urethritis, while gonococci are found in the discharges.

Before leaving the isolation-room, the patient should receive a cleansing, hot, soap-water bath (including thorough scrubbing of the scalp, ears, finger- and toe-nails), and dressed anew with freshly disinfected clothing.

2. Disinfection of Excreta, of Fomites, etc.—In order to be on the safe side, the nurse should be instructed to disinfect the stools, urine, vomitus, sputum, and nasal, aural, conjunctival and vaginal discharges of the patient, regardless of whether or not they carry contagious matter.

For Excreta.—Chloride of lime in powder or in solution. Four ounces of lime to one gallon of soft water. A sufficient quantity of this solution should be thoroughly mixed with the feces, urine, sputum, etc., and allowed to stand for about three hours before emptying.

Sputum is best collected in paper cups or small cloths and immediately destroyed by fire.

Disinfecting solutions.

Bichloride of mercury in solution 1 : 500—a 7½-grain tablet in a pint of water. Copper sulphate in solution (5 per cent.). Zinc sulphate in solution (10 per cent.). Cresol or creolin in solution (5 per cent.).

For Clothing, Bedding, Linen, etc.—Destruction by fire—the safest measure. Exposure to dry heat at a temperature of about 300° F., or moist heat at 212° F., for two hours. Boiling for at least half an hour. Immersion in a bichlorid solution (1 : 2000) for about three hours. Fumigation by formaldehyd (see below).

For the Hands, General Body, Dishes, etc.—Labarraque solution (chlorinated soda, 10 per cent.). Bichlorid of mercury in solution (1 : 1000). Permanganate of potash in solution (ℨj to a quart of water). Formaldehyd in solution (1 : 200).

Fumigation.

For Rooms, Furniture, Mattresses, etc.—Fumigation by Formaldehyd Gas.—It may be employed in concentrated powdered form or in pastels. For small rooms the ordinary Shering lamp, which is constructed for vaporizing formaldehyd pastels, will suffice. For large hospital wards, however, the *"formaldehyd-potassium-permanganate method"* is best. It is of advantage to use a container consisting of a large open vessel protected from losing its heat by some non-conducting material such as asbestos. But one can get along almost equally as well by using a large milk-pail set in a wooden bucket.

The infected room should be made as air-tight as possible by snugly closing the windows and doors (key-holes, ventilators, fire-places, etc.) by means of cotton or cloths. All articles intended for disinfection are freely exposed (mattresses, pillows, boxes and drawers should be opened).

Formaldehyd potassium permanganate.

The fumigating apparatus is placed in the center of the room; 6¾ ounces of potassium permanganate (for each 1000 cubic feet of room space) are put in the container; and 16 ounces of 40 per cent. formaldehyd solution (for each 1000 cubic feet of room space) are poured on the top of the permanganate. The operator now quickly leaves the room, and closes the door or window. The room should remain tightly closed for about ten hours.

After disinfection the disagreeable odor of the formaldehyd may be removed by sprinkling the room with ammonia water, and thorough ventilation.

Sulphur.

Fumigation with Sulphur.—The procedures are the same as with formaldehyd. The sulphur, about three pounds for a room 10 feet square, is placed in an iron pan, supported by bricks and set in a tin vessel with water. The sulphur is ignited by live coals or a tablespoonful of alcohol lighted by a match. Sulphur fumigation should not wholly be depended upon after grave epidemic affections.

Sunlight.

Finally, it is well to bear in mind that sunlight is a disinfectant of great efficiency, and that prolonged exposure to its rays will materially aid in rendering rooms and fomites free from infectious matter.

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III. IMMUNIZATION—ACQUIRED IMMUNITY. BIOLOGIC DIAGNOSIS AND THERAPEUTICS.

Common sense soon to reign supreme.

Medicine is rapidly reaching the goal of its highest ambition, the prevention and control of communicable diseases by "Nature's method," *i.e.*, immunization (see page 60). Stupid skepticism and boundless enthusiasm are gradually yielding to deliberate experimentation and experience, and it does not require a very great stretch of imagination to predict that in the near future every communicable affection will be successfully resisted and combated by an antagonist evolved by the causal microorganism.

In order to obviate unnecessary repetition we will briefly describe the biologic products at present in use for diagnostic, protective and therapeutic purposes and the results thus far achieved.

VARIOLA VACCINE.

Vaccinia.

With the enforcement of vaccination by all civilized nations small-pox, the most loathsome pestilence, has practically been eradicated from every well-regulated community. The principle of vaccination is the introduction into the human body of a weakened and harmless form of vaccinia, cow-pox, which renders the system immune (*i.e.*, creates enough of antibodies to resist the disease) to variola. The vaccine is obtained from the vesicles that form on healthy young heifers as a result of inoculation with the virus of cow-pox.

VACCINATION.

Aseptic precautions.

Signs of successful vaccination.

In the absence of contraindications (see page 94) every child of from 6 to 12 months old should be vaccinated, and revaccinated about seven years later. It is preferable to vaccinate at a time when neither excessive heat nor cold prevails, *i.e.*, in May or October. The right arm at the insertion of the deltoid is usually chosen for the first vaccination, and the left for revaccination. In girls the leg may be preferred to avoid the possibility of an exposed disfiguring scar. The parts to be inoculated should be freely bared and cleansed with soap-water and thoroughly dried. When one inoculation is to be made the epidermis should be abraded for about an eighth of an inch in diameter (until a serous exudate or a trace of blood occurs) by means of a sterile needle; when several inoculations are to be made, they should be fully one and a half inches apart. About a drop of vaccine is then gently rubbed into the denuded surface and allowed to dry. In successful vaccination the inoculated area begins to redden and swell on the third or fourth day; on the fifth day a vesicle appears which gradually changes into an umbilicated pustule surrounded by a red areola. The pustule persists up to the eleventh or thirteenth day and then becomes covered by a scab. The latter remains stationary about ten days longer, then falls off, leaving behind a red scar which gradually becomes white and glistening in appearance. The scar usually remains visible throughout life. Vaccination is associated with more or less marked constitutional symptoms. With appearance of the vesicle there is a slight rise of temperature; the child is restless, sleeps badly, loses its appetite, and shows other signs of indisposition.

Some children react more strongly than others, but if the vaccine is pure, the vaccinator clean and careful and the inoculated area kept free from irritation and infection, all the constitutional symptoms disappear by the twelfth day. Under adverse circumstances (*e.g.*, old, impure lymph, defective asepsis, constitutional diseases) vaccination may be accompanied by very grave symptoms. The pustules may become very large; the redness in the vicinity very marked and extensive; the axillary glands very much swollen and painful; the whole arm very strongly infiltrated; the fever very high, up to 104° F.; and convulsions and respiratory and gastrointestinal symptoms develop. Suppuration of the glands, phlegmonous processes, and even erysipelas may set in. Finally, vaccination may be accompanied by transient or genuine nephritis, and cases of scrofula, tuberculosis and syphilis are on record—undoubtedly pre-existent, latent, but awakened by the acute inflammatory process. Occasionally the inoculation wound fails to cicatrize, continues to suppurate or ulcerates. Children with a tendency to skin diseases may develop divers skin eruptions, such as erythema, eczema, lichen, impetigo, psoriasis, a purpura-like eruption (*purpura vaccinatoria*), general furunculosis, or, by transference (autoinoculation) of the vaccine virus to some diseased parts of the skin, produce general vaccinia. (The latter may develop—usually about the seventh or eighth day—spontaneously, from within, independently of any external influences. The lesions, which may be discrete or confluent (grave), bear a certain resemblance to the regular vaccinal pox.) In the same manner the vaccine may be carried to the eyes (*vaccine ophthalmia*), and cause serious trouble. In fact, inoculation pustules have been observed on different portions of the body, and even on the tongue. Furthermore, vaccinia may also be transmitted to other persons by means of infected articles in use, fingers, bed-sheets, bath-water, sponges, etc. Hence the importance of a protective dressing over the vaccination mark (clean sterilized linen, sewn to the sleeve, changed every day) from the time the vaccine has dried up to the falling off of the scab, and of keeping the child's nails very short and its hands very clean. Bathing should be interrupted from the fifth to fifteenth day. Moist boric acid dressings are useful to reduce the severe, local inflammatory process, and where the latter is grave, and the itching intense, a continuous, moist dressing with nitrate of silver (¼ per cent.)

Sepsis.

Awakening of latent tuberculosis, or syphilis.

General vaccinia.

Auto-inoculation.

Nitrate of silver.

will prove especially beneficial. In delayed healing the wound should be cauterized with a 5 per cent. to 10 per cent. solution of nitrate of silver, and dressed like any other wound. Other complications arising should be treated according to indications.

To guard against epidemics.

Revaccination.—As already suggested revaccination should be performed about seven years after the first vaccination, a period of time after which the immunity against small-pox usually ceases. In case of epidemics revaccination should be resorted to more frequently. Revaccination is also indicated to modify an attack of small-pox. In successful revaccination the local and systemic manifestations are essentially the same as after the first vaccination except that they are much milder in form.

Contraindications to Vaccination.—It is not advisable to vaccinate infants under three months, and children of all ages who are suffering from severe acute and recurrent skin affections, local or general syphilitic or tuberculous (scrofular) lesions and great debility.

ANTIDIPHTHERITIC SERUM.

Low mortality.

Diphtheria antitoxin is the purified blood-serum of a horse that has been rendered immune to diphtheria by a long course of treatment with diphtheria toxin. It is specific in its effects, having lowered the high (40 to 60 per cent.) mortality from diphtheria to about 5 per cent.—if administered early and in ample quantity. Furthermore, those exposed to diphtheria almost invariably escape infection by timely administration of the serum. It is practically harmless if free from admixture of virulent bacteria, and with introduction of the concentrated, high-grade preparations and the application of greater care in handling and administration, the numerous disagreeable accompaniments (fever, multifarious eruptions, articular swellings, etc.) have ceased to be as common and as severe as in former years.

Dosage.

The dose of antitoxin for ordinary cases of diphtheria should be 1000 units for every year of the child's age up to six years, to be repeated once or twice at intervals of from six to twelve hours. Malignant, especially laryngeal, cases require double doses. For protective purposes a third of the ordinary dose usually suffices. The protection usually lasts from four to six weeks.

The antitoxin is administered by a sterile hypodermic syringe (or the mercantile serum-containing syringes) by deep injection into the anterior surface of the abdomen or thorax or outer surface of the thigh, which are rendered aseptic by soap-water, ether and alcohol. The point of injection is subsequently sealed by sterile adhesive plaster.

Aseptic precautions.

ANTITETANIC SERUM.

Like diphtheria antitoxin, antitetanic serum is obtained from the blood of horses previously immunized to the toxin of the tetanus bacillus. Its efficacy as a curative remedy is as yet awaiting indisputable demonstration, but its value as a preventive of tetanus is authoritatively established. Whenever there is reason to fear tetanus infection (*e.g.,* contused or lacerated wounds—toy-pistol wounds—soiled with earth or other foreign matter) especially when an unusually large number of tetanus cases prevail, it is imperative promptly to administer tetanus antitoxin as a prophylactic measure.

Dosage.

Tetanus antitoxin is usually administered subcutaneously in doses of 1000 to 1500 units; the dose is repeated as a preventive measure after ten days, as a curative (3000 to 15,000 units) several times a day. In urgent cases the antitoxin may be given by intravenous, intracerebral or subarachnoid injection.

ANTIMENINGITIS SERUM (FLEXNER).

This serum acts specifically in cerebrospinal meningitis due to the diplococcus intracellularis (Weichselbaum) only. If used by the subdural method of injection in suitable doses, promptly and at proper intervals, it is capable of greatly diminishing the fatality generally due to the disease; of reducing the period of illness, and, in a large measure, of preventing the chronic lesions and types of the affection.

Dosage.

After reducing the intracerebrospinal pressure by withdrawal, by lumbar puncture (see page 339), of about 30 to 60 cubic centimeters of cerebrospinal fluid, we inject 30 cubic centimeters of the serum into the spinal canal by means of an antitoxin syringe or by gravity through a funnel and rubber tube attached to the puncture needle. The injection is repeated daily for three or four days or longer until the diplococci disappear. In fulminating cases a second dose may be given after the lapse of twelve hours. If after a period of apparent recovery the symptoms recur and the diplococci reappear, the injection should be repeated. The serum is practically useless in cerebrospinal meningitis after the condition of hydrocephalus has supervened.

Several other sera (*e.g.,* antipneumococcic, antidysenteric) are now on the market. Their curative merits, however, are still unestablished.

BACTERIAL VACCINES.

Following upon the great researches of our contemporary pathologists, bacteriologists and clinicians, A. E. Wright, of London, has demonstrated the remarkable fact that emulsions of dead bacteria—bacterial vaccines so called—if injected subcutaneously increase chemotaxis and, therefore, phagocytosis. The molecular group produced by the presence of the killed bacteria in the blood that renders the living bacteria of the same species a ready prey to the phagocytes he designated "opsonin," corresponding to the Greek verb "opsono"—I cater for, I prepare victuals for. He also devised a method to determine the "opsonic index," or sensitizing power of the blood, so that in a given case of infection one can, as it were, measure the opsonin content of the blood and increase it, if found below par.

Opsonic index.

Bacterial vaccine therapy is as yet limited to local infections, *e.g.*, furunculosis, phlegmons, carbuncles, where the offending micro-organisms can readily be determined by microscopic examination of the discharges, and accordingly the vaccine chosen to meet the indications.

Of the numerous vaccines thus far recommended the staphylococcus and streptococcus vaccines have actually stood the test and proved of great utility. They are deserving of more general application.

Strepto- and staphylococcus vaccines.

Favorable results are also on record from the use of vaccines prepared from the bacillus coli (in colicystitis); from gonococci (in gonorrheal affections, especially vulvovaginitis); from typhoid bacilli (in typhoid, especially as a preventive measure).

The inoculations are given by means of a sterile hypodermic syringe, in the same manner as antitoxin. In children particularly it is advisable to begin with small doses, let us say, 50 million staphylococci, or 2 million streptococci, and to increase the dose at each succeeding injection, which should occur every three to seven days.

Dosage.

In order to obtain prompt results it is essential to know not only the specific infecting micro-organism but also its variety, for instance, whether the offending staphylococcus is an aureus, albus, or citreus, as the employment of a different variety of vaccine is apt to prove useless.

Bacterial vaccines are often prepared directly from cultures obtained from the individual to be treated.

TUBERCULINS.

These bacterial products are invaluable in the early diagnosis of tuberculosis in children. By means of tuberculin we are enabled to detect from 90 to 95 per cent. of cases of tuberculosis, often at a time when no other clinical manifestations or bacteriologic examinations indicate its presence. It has furthermore the great advantage that its use calls for no complicated procedures, methods, calculations or instruments. The specific test is based upon the fact that on meeting with the antibodies evolved by the organism the tuberculin sets up a reaction, which is manifested either by a local inflammation or systemic disturbance.

Specific tuberculin tests.

The tuberculin reaction may be elicited in the following manner:—

1. **The Cutaneous Method (von Pirquet).**—After cleansing the anterior surface of the forearm with soap-water and ether, two small abrasions (as for vaccination) or punctures of the skin are made at an interspace of about two inches. On one of the two abraded spots a drop of a 50 to 100 per cent. solution of Tb is applied and allowed to dry. If tuberculosis is present, a red pea- to bean-sized papule appears after from twenty-four to forty-eight hours at the point of contact of the injured skin and tuberculin, while the other non-tuberculized spot remains free from the inflammatory reaction.

Red papule.

2. **Conjunctival Method (Calmette).** A drop of ½ to 1 per cent. (trying the weaker solution first) of old Tb solution is instilled into the conjunctival sac of one eye. In the presence of tuberculosis a positive reaction is manifested within twenty-four hours by reddening of the caruncles and semilunar fold of the conjunctiva and injection of the corneal conjunctiva. The other eye remains normal.

Congestion.

3. **Nasal Method (Wolff-Eisner and Calmette).**—A cotton tampon saturated with a 1 per cent. solution is applied against the nasal septum and allowed to remain there for about ten

Yellow crust.

minutes. In from eighteen to forty-eight hours a peculiar exudation appears which dries and forms a yellow crust upon a congested mucosa. From this clumps of extravasated red cells project here and there as minute reddish points. The crust generally falls off from the fourth to the sixth day.

4. Percutaneous Method (Moro).—This method is less reliable than the aforementioned procedures. A 50 per cent. tuberculin ointment is rubbed over about a square inch of epidermis until absorbed. If the reaction is positive, papules appear within from twenty-four to forty-eight hours.

Papules.

5. Subcutaneous Method.—Almost never employed in young children.

Tuberculin-therapy.—A very enthusiastic revival has recently taken place in the employment of tuberculin in the treatment of tuberculosis, especially of bones, joints, glands and the skin. As during the period of the tuberculin treatment the patients are receiving also the benefits of outdoor air, good food, tonics, etc., it is still questionable whether the results warrant the unbounded enthusiasm. However, the administration of tuberculin in minute, gradually increasing doses ($\frac{2}{10}$ mg., $\frac{3}{10}$ mg., $\frac{5}{10}$ gm., 1 mg., etc., every three days, up to 1 cg. or more[1]—subcutaneously into the cellular tissues of the thorax) being harmless, there is no objection to its use in selected cases.

SERUM DIAGNOSIS OF SYPHILIS (WASSERMANN).[2]

The substances employed in this reaction are as follows[3]:—

Mode of preparation.

1. Fresh Serum of the Guinea-pig.—The animal is bled from the carotid or the femoral artery. The blood thus obtained is either rapidly centrifuged or allowed to stand for some time after first removing the upper layer of the clot which adheres to the walls of the receptacle. The separated serum floating over the clot is drawn off by means of a pipette with a rubber tube and glass mouthpiece attachments. The serum should be kept on ice ready for use, and before using diluted with ten parts of salt solution.

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[1] Centralbl. f. Kinderheilkunde, May, 1910.

[2] In view of its comparative simplicity, the technique perfected by Dr. J. Bauer, of Düsseldorf (La tribune méd.), is here described.

[3] Noguchi employs for Wassermann's reaction small squares of paper representing measured amounts of the antigen, amboceptor, and the complement, thus greatly simplifying the method and enabling the physician to perform the test in his office, provided he can procure active Noguchi test-papers.

2. **Washed Sheep's Blood-corpuscles.**—This is a 5 per cent. suspension. The blood is collected from the jugular vein of a sheep in a sterile bottle containing iron filings, to avoid coagulation, and is shaken for ten minutes. It is then strained through a sieve to remove the fibrin and centrifuged and washed with salt solution. This is repeated several times until the solution over the sediment remains quite clear. The liquid is then poured off and an equal (the same as it was after the first straining) quantity of normal salt solution is added instead, so that the proportion of the blood-corpuscles remains the same. From this suspension we prepare a 5 per cent. solution (by adding to one part of the blood mixture twenty parts of physiologic salt solution) and place it on ice ready for use.

3. **Normal Human Serum.**—This is obtained preferably from the blood of a placenta. The serum should be heated over a water bath up to about 130° F. to render it inactive.

4. **The Syphilitic Extract.**—This is prepared by triturating in a mortar 100 cubic centimeters of alcohol (96 per cent.) and 10 grammes of the liver of a syphilitic infant, allowing it to stand (well covered) over night, centrifuging, decanting the clear liquid and placing it on ice.

Experimental test.

With the mother solution of the organic extract ready, we now proceed with an *experimental* test as follows: Into a series of test-tubes we pour, respectively, 0.25, 0.15, 0.10, 0.05, 0.025 and 0.015 cubic centimeter of the mother solution of liver extract, and to each of the tubes we add enough of physiologic salt solution to make its total content equal 1 cubic centimeter. In addition another test-tube (control tube) is filled with 1 cubic centimeter of salt solution without any organic extract. In each of the six tubes containing the extract we next pour 1 cubic centimeter of the fresh 10 per cent. solution of guinea-pig's serum, then 0.2 cubic centimeter of normal human serum heated up to about 130° F. The series of tubes is next placed in an incubator at 99° F. for thirty minutes, then each tube charged with 1 cubic centimeter of a 5 per cent. of sheep's blood-cells, and again put into the incubator for two hours. If now the contents of all the tubes (except, of course, the control-tube, in which the liquid should always be clear) are found dissolved, the first tube can be used, otherwise any of the remaining tubes in which the solution is complete. As each tube contains 1 cubic centimeter of fluid and indicates the amount of organic extract therein, we can readily tell how much of the latter is required for the diagnosis. Suppose, for example, that in the experiment the fourth tube be selected as perfectly dissolved, we at once know that the quantity of organic extract needed for the test is 1 in 20. The correctness of the conclusion should be verified by repeating the test with different dilutions of the extract (1 : 10, 1 : 20, 1 : 30, etc) and several specimens of blood of healthy and positively syphilitic persons.

5. The Serum of the Patient.—The serum is obtained by puncturing a subcutaneous vein or finger with a large needle and collecting it in a test-tube. Allow it to clot; remove the separated serum; centrifuge to clearness; pipette off into another test-tube, and render it inactive by an half-hour's exposure to 130° F.

Actual test.

Now that everything is ready for the *actual* test, we fill four test-tubes as follows:—

Tube 1.

The patient's serum	0.2 c.c.
The organic extract (tested)	1.0 c.c.
The guinea-pig's serum (1:10)	1.0 c.c.

Tube 2.

The patient's serum	0.2 c.c.
Physiologic salt solution	1.0 c.c.
Guinea-pig's serum (1:10)	1.0 c.c.

Tube 3.

Normal blood-serum	0.2 c.c.
The organic extract (tested)	1.0 c.c.
Guinea-pig's serum (1: 10)	1.0 c.c.

Tube 4.

Normal blood-serum	0.2 c.c.
Physiologic salt solution	1.0 c.c.
Guinea-pig's serum (1: 10)	1.0 c.c.

The last three tubes serve merely for comparison to make sure that there are no accidental errors which render the test unreliable. The four tubes are shaken and placed for thirty minutes in the incubator at 99° F. To each then is added 1 cubic centimeter of the 5 per cent. suspension of sheep's blood-cells and the reaction is then watched in the incubator.

Usually in tubes 2 and 4 the contents become clear in from fifteen to thirty minutes. Hemolysis then appears in tube 3. When the blood-corpuscles in tube 1 dissolve almost simultaneously with those in tube 3, the patient is *free from syphilis.* On the other hand, if the contents of tube 1 do not dissolve, the suspected patient *has syphilis.* The test is of no value unless the contents of tube 2 completely dissolve. If the contents of tube 1 dissolve imperfectly about half an hour or so after hemolysis in tubes 2 and 3, the existence of syphilis is possible, and therefore we must start the test again with tubes 1 and 2, but with smaller quantities of the patient's serum, *e.g.*, 0.15 cubic centimeter. If this is not successful, we repeat the test with 0.1 cubic centimeter and again with 0.05 cubic centimeter of the patient's serum, endeavoring to find that combination which will allow the contents of tube 1 to remain intact while those of tube 2 to dissolve completely. If this is obtained the diagnosis of syphilis is still fairly certain.

Negative.

Positive.

If the contents of tube 2 do not dissolve completely, we should add to tubes 1 and 2 from 0.1 to 0.2 cubic centimeter of human normal serum from fifteen to thirty minutes after (to make sure that the contents of tube 2 do not dissolve) the addition of the 5 per cent. solution of the sheep's blood suspension. The idea is to find for tube 2 the amount of normal human serum that will exactly dissolve its contents, and then to use the same amount for tube 1.

Judging by the conclusions arrived at by different clinicians, the serum reaction for syphilis is specific and found positive in from 90 to 95 per cent. of all cases with syphilitic manifestations. It is invaluable, especially in the detection of latent forms of the disease.

SERUM DIAGNOSIS OF TYPHOID.

(Grüber-Widal.)

The blood of persons suffering from typhoid, when added to a broth culture of typhoid bacilli, arrests the characteristic movements of these germs and produces their agglutination and sedimentation. This phenomenon may be observed macroscopically in a suspension of bacteria in test tubes; or, microscopically, when the bacteria are mixed with the blood and mounted in a hanging drop preparation. The test is generally positive in typhoid patients after the fifth day of the disease and several weeks thereafter.

Positive after fifth day.

The blood (or serum from a blister) is obtained from the skin covering the ear lobe. After cleaning this part, the lobe is pricked with a sterile needle, and two drops of blood are placed on a glass slide, one near either end and allowed to dry in the air. The examination can then be undertaken any time thereafter by diluting one drop of the blood in ten or twenty parts of the typhoid culture.

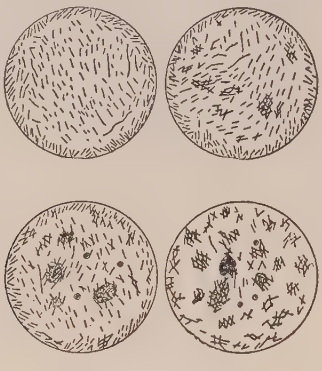

Fig. 27.—Stages in Widal Reaction. (After *Robin.*)

IV. MATERIA MEDICA AND THERAPEUTICS.

(Including Hydrotherapy, Electricity, Massage, Climatotherapy and Organotherapy.)

No one method of treatment suits all cases. Some diseases subside spontaneously, if left alone; others go from bad to worse if not treated promptly and energetically. Some affections yield readily to biologic remedies, others to crude drugs or synthetic pharmaceutical preparations, and again others respond to change of climate, mode of living and eating, and to remedial measures other than pharmaceutical, such as hydrotherapy, massage, electricity and the like.

Our duty being to alleviate suffering, we owe it to our patients to keep pace with the advances of the time and to employ every useful method of treatment regardless of its source or character. "The period of exclusiveness is past." While a certain degree of conservatism is always wise and safe, skepticism to well-tried remedies is worse than folly.

Conservatism versus nihilism.

HYDROTHERAPY.

The virtue of water as a therapeutic agent varies with the idiosyncrasy of the patient, the temperature of the water employed, and the method of its application.

Heat applied to the surface of the body produces a relaxation of the vasomotor system. The cutaneous vessels dilate and become more active, diaphoresis ensues, and effete matter is eliminated. The volume of blood in the deeper structures is diminished; hence, congestion relieved. The temperature of the body is first increased, but after free diaphoresis considerably lowered.

Dilatation of cutaneous blood-vessels.

Cold contracts the terminal blood-vessels and stimulates the internal circulation. It reduces the temperature of the body not only by conduction but also by inhibition of heat production. Soon after discontinuance of the cold a reaction takes place, respiration becomes deep and full, more carbon dioxid is excreted and the supply of oxygen is increased. The pulse, which is at first feeble, soon becomes full and strong; the chilliness and rigor disappear, and a sensation of warmth pervades the body surface. The blood-current in the capillaries becomes gradually accelerated and the internal circulation relieved of its tension.

Contraction of terminal blood-vessels.

The External Use of Water.—Neither extreme heat nor extreme cold should be employed in the treatment of diseases of children. Heat should be avoided on account of the severe depression, and cold because of the shock it is apt to produce.

Cold Sponging.—In the employment of cold water in the treatment of diseases of children, sponging advantageously supplants the cold bath. The temperature of the water should vary between 70° and 90° F. Three basins of water, one each of 70° F., 80° F. and 90° F., respectively, are placed at the bedside. The child is stripped and laid upon a blanket, and by means of cloths the surface of the body is sponged for from two to three minutes, in the following order of succession: face, neck, chest, back, abdomen, buttocks, upper and lower extremities. The warmest water (90° F.) is used first and the coldest (70° F.) last. Each part of the body should be thoroughly dried immediately after it has been sponged. The indications for the use of the sponge bath are hyperpyrexia and nervous irritability; constitutional disorders, such as anemia chlorosis, scrofula, etc., and in cases in which a general tonic effect is desired. In the latter conditions sponging should be followed by active friction.

Antipyretic.

Cold Wet Pack.—The child is stripped and blankets are placed over and under it. A small sheet is dipped in water at a temperature of 70° to 90° F., thoroughly wrung out and wrapped loosely around the patient. The child's body is then enveloped in the blankets. To reduce high temperatures, for example, in typhoid or pneumonia, ice may be rubbed over the chest. The next pack is applied after an interval of ten minutes and may be repeated from ten to twelve times in twenty-four hours. The feet should be kept warm by artificial heat.

In nephritis.

Vapor Pack.—If the cool wet pack is allowed to remain in position for from one to two hours and loss of body heat prevented by thoroughly covering the child with woolen blankets, the cold pack is converted into a warm pack which produces effects similar to those obtained from a vapor bath; namely, free diaphoresis, lowered activity of the nervous system, calm and repose, and equalization of the internal circulation. The vapor pack is, therefore, invaluable in acute catarrhal conditions of the air passages, in nephritis, dropsical effusions, muscular rheumatism, eclampsia, hyperesthesias, etc.

In local inflammations.

Wet Local Compresses (Priessnitz)—*Cold Compresses.*—These are applied in all forms of local inflammation, to relieve pain, swelling, heat and redness. In order to obtain good results, the temperature of the water should vary between 50° and 60° F., and the compress left in place and kept cold either by frequently sprinkling cold water over it or by the application of an ice-bag. Indications: Meningitis, angina, acute pharyngitis and laryngitis, hemoptysis, appendicitis, intestinal hemorrhage, etc.

Warm Compresses.—While cold compresses delay the flow of blood and cell-activity, warm compresses accelerate the blood-current and promote cell-activity. They are applied by means of cloths immersed in water at a temperature of about 100° F., thoroughly wrung out, and then covered with flannel and rubber tissue or oiled silk to prevent rapid evaporation and cooling. The compresses should be changed as soon as they become dry.

In local pain and spasm.

Indications: Neuralgia of the head; throat affections after subsidence of the acute inflammatory stage, to promote absorption of diseased products; in exudative pleuritis; in bronchitis, to allay severe cough and to promote expectoration; in all spasmodic conditions of the intestines; to hasten suppuration and relieve stasis.

In eruptive fevers.

Baths.—*Tepid Bath.*—This is a very useful bath in children. The temperature of the tepid bath varies between 85° F. and 92° F. It is employed in diseased conditions requiring soothing, for example, in eruptive skin diseases and as an antipyretic in infectious diseases.

Warm Bath.—In a general sense, this is the most valuable bath in the treatment of diseases of children. It tranquilizes the nervous system, equalizes the circulation, produces diaphoresis and reduces temperature.

Nerve sedative.

Indications: All spasmodic conditions; affections of the lungs and kidneys; exanthematous diseases, and nervous affections, such as hysteria, etc. The temperature of the bath should vary between 92° F. and 98° F. The patient should remain in the bath for from two to five minutes. The warm bath is sometimes employed as a *permanent bath,* in extensive burns and wounds, and in skin diseases associated with intense itching. The patient is suspended in the bath on a sheet. The water is kept at an equal temperature by proper arrangement of inflow and outflow.

In collapse; convulsions.

Hot Bath.—The temperature of the hot bath may be carried as high as 108° F., and the patient should remain in the bath for from one to three minutes. It is very useful in collapse, convulsions and chronic rheumatic conditions. It is occasionally administered to break up a "cold," and to produce rapid diaphoresis. While in the bath the patient's head should be kept cool by an ice-bag.

Nerve stimulant.

Shower Bath.—Cold shower baths are generally given for their stimulating effect. Hence, they are of great value in nervous affections, such as neurasthesia; in enuresis, and as a general tonic. For these purposes one shower (shock) at a time is sufficient. The shower bath should be followed by active friction.

Local stimulant.

Aspersion Bath.—The value of cold water dashed suddenly over the frame or directed in a steady, broad stream upon some particular part, is very great. The cases in which such a mode of treatment is beneficial are numerous. The following are a few of the more important: Where the muscular power of a leg or arm is impaired from long inaction, as in cases of fracture, dislocation, bandaging, sprains and partial paralysis. The patient sits in a bath-tub or on the floor and the operator, standing on a table, directs the stream of cold water upon the affected part from a watering-can from which the sprinkler has been removed. This mode of treatment is rendered particularly serviceable if the circulation is quickly restored by vigorous dry friction for several minutes. It is also efficacious in systemic poisoning from drugs, suffocation from noxious gases, etc.

Medicated Baths.—Aside from the natural mineral baths obtained in the celebrated spas, which will be discussed later, a number of artificial baths are commonly used in the treatment of diseases of infancy and childhood. The efficacy of these baths is, in the majority of instances, due probably to the effects of heat or cold and friction employed with the non-medicated bath.

Stimulant.

Aromatic Bath.—About six ounces each of chamomile flowers, calamus roots and peppermint leaves are tied up in a muslin bag and thrown into a warm bath. Aromatic baths are recommended in marasmus, infantile spinal and other forms of paralysis, in sclerema, etc.

In skin diseases.

Bran Bath.—Two or three pounds of wheat bran are boiled for about an hour in about three quarts of water. The decanted liquid is added to the bath. It is useful in intertrigo, eczema, pemphigus, lichen, strophulus, etc.

Malt Bath.—A few ounces of malt extract are added to the bath. Malt baths are recommended in rachitis, spasm of the glottis, and in general debility.

In syphilis.

Mercurial Bath.—This form of bath is employed as an adjuvant in the treatment of syphilis. It is usually prepared by the addition of 20 to 30 grains of calomel, or 0.5 to 1.0 gramme (gr. viiss to xv) of bichlorid of mercury.

To break up "cold."

Mustard Bath.—Two or three ounces of mustard are dissolved in a few pints of tepid water and added to the bath. The temperature of the bath may vary between 100° F. to 106° F. It may be administered in the form of a sitz bath or full bath. The patient should remain in the bath for from ~~three to~~ ten minutes. Mustard baths are indicated in collapse, shock or heart-failure from any cause, in sudden congestion of the lungs or brain, etc.

Stimulant.

Sea-salt Bath.—About two pounds of sea-salt are dissolved in the bath of four or five gallons of water. It is stimulating in its effects, and useful in rachitis, various forms of paralysis, etc.

In skin diseases.

Soap Bath.—This form of bath is employed in the treatment of prurigo, lichen, strophulus, scabies, etc. It is prepared by the addition of from three to six ounces of soft green soap to five gallons of water.

Sulphur Bath.—A half to one ounce of potassium sulphuret should be added to each bath. In some cases the addition of about three ounces of animal gelatin is of advantage. Sulphur baths are deserving of recommendation in rheumatism, eczema, prurigo, urticaria, lead poisoning, etc.

In skin diseases.

The Internal Use of Water.—The benefits derived from the internal use of water are manifold, but unfortunately greatly underestimated. Water taken by the mouth in moderate quantities—large amounts weaken digestion—cleanses the alimentary canal, stimulates peristalsis and produces diuresis and diaphoresis. To a certain extent it acts also as a food. In acute diseases associated with anorexia the free use of water will often sustain life for weeks. In febrile diseases water not only quenches thirst, but aids also in the reduction of temperature. Water stimulates expectoration, and in the form of cracked ice checks vomiting. For the latter purpose small sips of hot water are sometimes resorted to.

Diuretic.

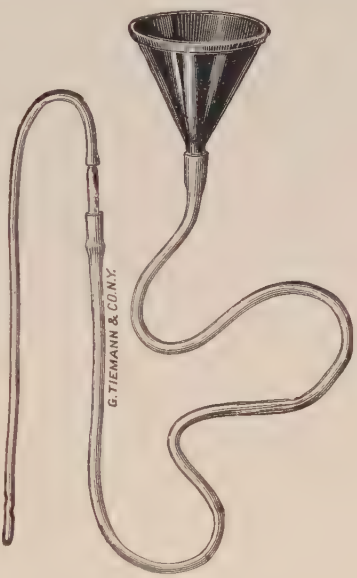

Fig. 28.—Stomach Tube (catheter, rubber tube, glass connection, and funnel).

Lavage.

Lavage.—Stomach washing in children is performed in the same manner as in adults. Its field of usefulness, however, is much wider. It is invaluable in cases of acute simple and toxic gastritis, cholera infantum, chronic indigestion and difficult feeding. A funnel with a few feet of rubber tubing, to which a small soft-rubber catheter (No. 12 or 14) is joined by means of a glass cannula, is the best apparatus for stomach washing. About ten inches of the catheter should be passed beyond the lips. The temperature of the irrigating solution should be about 100° F., or higher, if special indications arise. The quantity of solution to be instilled varies with the capacity of the child's stomach. Generally, pure boiled water answers all medicinal purposes, except in poisoning, in which instance antidotes may be employed. In hyperacidity of the stomach bicarbonate of soda or lime-water may be added. Lavage is contraindicated in heart disease and hemorrhagic diathesis.

To cleanse stomach.

Contra-indications.

Irrigations.—The action of irrigations is chiefly mechanical. They are indispensable in the treatment of divers affections of the lining membranes of internal cavities. In chronic cystitis, for example, washing of the *bladder* by means of sterile or medicated (boric acid, silver nitrate) water will often rapidly effect a cure.

To cleanse internal cavities.

Irrigations of the *vagina* are frequently employed in vulvo-vaginitis. A slow current of water should be employed, permitting the fluid to return without injury to the adjacent parts. A fountain syringe with a small, sterile, soft-rubber catheter attached, generally suffices for ordinary purposes. The water-bag should be suspended about two feet above the child's body.

Irrigations with warm, sterile water are very beneficial in *ear affections,* such as impacted cerumen, foreign bodies in the external auditory meatus and external otitis.

In febrile diseases, adenoids, chronic pharyngitis, etc., instillations of weak salt water or ichthyol solutions prevent and cure affections of the nasopharynx and ear; it often also relieves reflex cough and embarrassed respiration. Instillation may be performed by means of a teaspoon or dropper, and should be repeated at least twice a day.

Copious irrigations of the *mouth* with sterile or medicated (silver nitrate, hydrogen peroxid) water are invaluable in the treatment of grave forms of stomatitis.

Enteroclysis.—The indications for *low enemas* are too well known to need further discussion. It may be mentioned, however, that in habitual constipation only small quantities of water should be injected into the bowel. Large quantities are apt to produce atony of the colon by overdistention and thus aggravate the disease.

High enemas are given by means of a flexible (colon) tube and a fountain syringe. High enemas not only remove effete material from the intestines, but by using water at a temperature of 80° to 90° F. also reduce temperature. Hence, they combine two therapeutic measures, which are of signal benefit in all gastrointestinal disorders, peritonitis, typhoid, etc. Soap-suds, turpentine, starch and salt, among other adjuvants, may be added according to indications.

Cleanser and stimulant.

Saline injections stimulate the kidneys and promote elimination of putrid material. They stimulate the circulation and supply the deficiency of body fluids in conditions associated with an excessive drain of fluids. Saline injections are, therefore, a sovereign remedy in uremia, typhoid fever, scarlet fever, smallpox, measles, diphtheria, eclampsia, anemia, hemorrhages, and in shock after surgical operations, etc.

General stimulant.

A physiological (0.9 per cent.) salt-water solution at a temperature of from 100° to 110° F. is generally used. It should be injected slowly through a colon tube, and continued for from fifteen to twenty minutes.

Saline injections are contraindicated in chronic kidney disease.

Hypodermoclysis.—Subcutaneous injection of salt water (110° F.) is performed by means of an ordinary fountain syringe with an antitoxin syringe-needle attached. The needle and skin should be rendered aseptic. The injection should be made in places where there is an abundance of subcutaneous cellular tissue, for example, the anterior surface of the abdomen and thorax. The current should be very slow, and the quantity of the saline solution to be injected should vary between from two to six ounces, according to age and indications. Hypodermoclysis is of inestimable value in cases of collapse resulting from hemorrhage; in pneumonia; uremia; acute gastroenteritis with great loss of body fluids; and in leukemia. It should be preferred to intravenous infusion.

In collapse, especially from loss of blood.

ELECTRICITY.

Electricity as a remedial agent in the treatment of diseases of children is employed in the following forms, in the order in which they are named: Galvanic, Faradic and Static.

Nerve and muscle stimulant.

The Galvanic Current.—The effect of the galvanic or direct current on the *muscle* is to produce contraction. The contraction takes place at the moment the current is closed or opened (make or break). The galvanic current, if applied by means of two electrodes along the course of a *motor nerve,* produces a uniform contraction of the entire muscle supplied by that nerve. The reaction produced by the constant current upon the *sensory nerve* varies according as the application is made with the positive or negative electrode, the anode being sedative in its effects, the cathode stimulating. A constant current of suitable strength—10 to 15 milliampères—passed through living tissues causes, at the point of contact of the anode, an accumulation of oxygen, chlorin and acid; coagulation and shrinking of the exposed tissue—*positive electrolysis*. On the other hand, if the cathode is brought in contact with living anmial tissue, hydrogen and the alkalies are set free, and liquefaction of the parts adjacent to the electrode takes place—*negative electrolysis.*

Nerve and muscle stimulant.

The Faradic Current.—The faradic or induced current causes contraction of muscles and nerves and is very effective in producing muscular massage. It stimulates nerve action and nutrition, excites secretion, and arouses latent physiological function.

Tonic and sedative.

The Static Current.—The static current produces vivid and persistent contraction of a large group of muscles with a minimum of pain. The second prominent characteristic of this current is its power of relieving pain.

The following rules should be borne in mind:—

1. Always administer the weakest possible current that will cause muscular contraction.

2. Never employ electricity in the inflammatory stage of organic disease.

3. In applying electricity to muscles always endeavor to separately reach the electromotor points. In deep-seated muscles the current should be applied along the course of the nerves supplying them.

4. Each electric treatment should last no longer than twenty minutes, and no one muscle should be subjected to the currents for more than three minutes.

The indications for electricity in the treatment of diseases of children are practically the same as in adults. The discussion of the subject will, therefore, be limited to diseases in which electricity is of undoubted value.

Chronic Constipation.—The galvanic or faradic current may be used. One electrode is passed successively over different portions of the abdominal wall, and the other electrode is placed upon any other part of the body. The electric treatment should be continued for a long period.

Diphtheritic Paralysis.—In this condition, faradization of the respiratory muscles, particularly the diaphragm, is of some service. It should be used in attacks of respiratory failure and continued while they last.

Enuresis.—The broad anode is placed over the lumbar region of the spine and the small cathode over the region of the bladder or upon the perineum, allowing quite a strong galvanic current to act for from two to four minutes. Sometimes faradization proves effective. The wire end of a conducting cord, connected with the negative pole, should be introduced into the urethral orifice for from one to two centimeters and quite a strong faradic current allowed to act for from one to two minutes.

Value of electricity in different diseases.

Facial Paralysis.—This form of paralysis is greatly benefited by a weak stabile galvanic current. It should be employed four to six times a week, for from two to three minutes at a time. The anode should be placed in the auricular fossa and the cathode upon the muscles of the affected side; or the anode may be placed behind the ear while the different nerve branches and the muscles are slowly stroked with the cathode. In later stages faradization also is of service.

Hysteria.—The vague disconnected symptoms of hysteria call for general electric treatment, and no form of electricity so advantageously combines tonic and sedative effects as the static current. A mild current should be employed. Two or three treatments a week will generally suffice. Galvanism and faradism also are of service, especially in hysterical contractures.

Multiple Neuritis.—The application of electricity to the affected muscles is important in order to maintain their nutrition. It should be begun after the acute stage has passed, that is, at the end of from three to four weeks. A moderate faradic current may be used if the muscles respond to it; otherwise a voltaic. The electricity should be applied daily by means of large electrodes, so that the current may reach as much muscular tissue as possible. The current should be strong enough to produce visible contraction of the muscles.

Poliomyelitis.—The galvanic current gives the best results. It should not be employed earlier than the third or fourth week. A large, flat electrode, well moistened in salt water, is placed upon the spine over the affected region and the muscles repeatedly stroked by means of a small electrode. The current should be of such strength as will produce visible contraction of the muscles, without, however, causing severe pain to distress the child.

Rheumatism.—The sequelæ of rheumatism, atrophy and contractures, often call for electric treatment. The galvanic, faradic or static current may be employed. It is sometimes advantageous to use the galvanic and faradic currents at one sitting. The treatment should be repeated at least every alternate day and continued for several months. In muscular contracture the anode should be placed over the portion of the spine governing the contracted muscles and the cathode over the muscles themselves. For the relief of pain the positive pole should be applied to the most painful spot.

Tetany.—Electric treatment has been followed by improvement in a number of cases. The stabile galvanic current should be employed; the negative pole to the spine and the positive to the irritable nerve trunks.

Torticollis.—A weak galvanic current is frequently very serviceable. The positive pole should be placed just below the occiput and the negative pole allowed to act upon the contracted muscles for from five to ten minutes.

The indications for electrolysis are identical with those in adults.

MASSAGE.

Massage is a mechanical form of treatment consisting of intelligent manipulations of the superficial parts of the body. It is intended to produce changes in the local and general nutrition, action and other functions of the body.

Stimulant.

To remove local tumefactions.

Indications.—Massage is indicated in hysterical, paralytic, rheumatic and traumatic contractures of joints; in fractures, to hasten absorption of callous masses; in chronic glandular enlargements; in swellings associated with rheumatism, sprains, contusion, etc.; in torticollis, to relax muscular contraction; in constipation, atonic dyspepsia and gastric dilatation; in all forms of muscular atrophy or dystrophy; as a general stimulant in cases of prolonged muscular inactivity, whether from indolence, disease, feebleness (rachitis), or prolonged use of splints or braces, or other cause; in various forms of paralysis, to improve the nutrition and function of the affected muscles.

Contraindications.—Massage is contraindicated in children suffering from gonorrheal rheumatism or peliosis rheumatica; in tuberculous, typhoid or syphilitic ulcerations of the intestines; in acute peritonitis, appendicitis, gastroenteritis, gastric ulcer; in tubercular glandular enlargements.

Massage is generally divided into the following principal manipulations:—

Effleurage or Stroking.—In making the strokes both hands are employed. The limb is grasped with one hand just above the other, in such a manner that pressure is exerted to some extent

Methods of application.

by the whole palm, but especially the ball of the thumb and the inner surface of the last two phalanges of the fingers. The strokes are delivered in the form of an ascending spiral, the two hands being moved simultaneously in opposite directions, the lower following closely upon the upper. The strokes must be made with great regularity. Light stroking has a soothing influence; heavy stroking stimulates the superficial structures increasing the arterial, venous and lymphatic circulation.

Friction.—This manipulation is performed with the finger-tips and consists of firm circular, semicircular, or to and fro movements. It is usually combined with effleurage and is intended to promote absorption by the veins and lymphatics.

Petrissage or Kneading and Pinching.—In kneading the endeavor of the operator is to pick up the individual muscle or muscle-groups between the fingers of the two hands, or in some cases between the thumb and finger of one hand, and then to roll and squeeze the muscle with a double movement. These manipulations cause circulatory, nutritive and alterative changes in the muscles, tendons and organs within reach.

Tapotement, Percussion or Tapping.—Percussion is made either with the points of the fingers brought into a line with one another or with the side of the hand and fingers. The movement should be very rapid and elastic. These manipulations are usually employed on muscular parts, such as the back of legs and gluteal regions. The effect of tapotement is similar to that obtained by petrissage. This manipulation may be enforced also by vibrations, that is, by rhythmic, tremulous movements under pressure.

Gentle manipulation.

Generally, all the movements are practised at one sitting, thus, effleurage, friction, petrissage, tapotement and vibration. The treatment is concluded by effleurage. While in local affections local massage is generally sufficient to effect the desired results, it is always advantageous to supplement the local treatment by general massage. The duration of each séance varies from a few minutes to a quarter of an hour. At first the treatment should not last more than five minutes. No force should be used, and the delicate skin of the child should be spared unnecessary injury. It is, therefore, advisable to anoint the skin with boric acid vaselin, cocoanut oil or any other emollient. In young infants massage should be limited to general friction of the body. In cases of malnutrition it is a good rule to give a fat-inunction daily after the morning bath.

CLIMATOTHERAPY.

Change of climate has from time immemorial been recognized as a therapeutic measure *par excellence,* and, fortunately, our great country abounds with vast mountain, seashore and inland resorts, which rival, if not surpass, the most celebrated spas of Europe.

Importance of individualization.

In selecting a suitable health resort, we should bear in mind not only the state of health and the peculiarities of the individual patient, but also the local conditions of the particular resort, such as the drainage, water supply, prevalence of epidemic or endemic diseases, etc.

Mountains.

The air of mountainous regions is rarefied, dry, cool, bracing and free from organic and inorganic impurities. It improves the action of the skin; favors deeper expansion of the lungs, and correspondingly accelerates the heart's action, improves sleep and stimulates the appetite and the powers of assimilation. Mountain air, therefore, is particularly beneficial in chronic disorders of the alimentary tract and liver; in anemia; in divers respiratory affections; in malaria; in rheumatism, and compensating heart disease.

Seashore.

The climate of the seashore is pure and very strong. The air is loaded with moisture, and comparatively free from dust particles, hence very beneficial to convalescents from pneumonia, pleurisy and empyema; also typhoid and surgical operations. It often acts almost specifically in acute gastroenteritis.

The surf-baths are invaluable in cases of nervousness, rachitis and local tuberculosis.

Inland resorts.

Dry, sheltered inland resorts are to be preferred for patients suffering from non-compensating heart disease; severe bronchitis; chronic kidney disease, and all such affections as are apt to be badly influenced by sudden variations of temperature.

It is often of advantage to spend part of the summer months at the seashore and part time in the mountains or inland resorts. Young children suffering from tuberculosis will, during the winter months, derive the greatest benefit from a sojourn in New Mexico and Arizona. Children over ten years old often do well in colder climates, such as the Adirondacks.

MATERIA MEDICA.

As already suggested, quite frequently we have to resort to drugs for the relief and cure of the patient. It is very unfortunate that medical students are nowadays given so little opportunity to familiarize themselves with the intrinsic value of a great number of old and new pharmaceutical products. I am firmly convinced that so-called "Medicinal Nihilism," to a great extent, is due to lack of knowledge of the physiologic action of the numerous standard drugs and inexperience as to their indications and mode of administration.

Lack of knowledge responsible for medicinal skepticism.

Palatable Medication.

Palatable medication is, to say the least, highly appreciated by sick adults, and practically indispensable in the management of sick children. The physician who believes in the usefulness of the medicines he prescribes owes it to his patients that they are able to swallow and retain them. As a rule, adults manage, by means of condiments and pleasant beverages, to render drugs disgusting in taste at least acceptable. On the other hand, children are compelled to take the medicine as given to them, and what is still worse, the more they resist the more they are subjected to anguish and distress, nay, even to corporal punishment which not infrequently borders on actual injury. Indeed, it is not at all rare to find children suffering from acute pneumonia in a state nigh to suffocation from the effects of prolonged and firm compression of the nostrils; and many a child bleeds from gums and lips, and loses a tooth or two through the attempts of the overzealous mother to force down into the unfortunate's throat a teaspoonful of a miserable mixture—which was, perhaps, intended only as a placebo.

Forced drugging dangerous.

As most drugs are now obtainable as solid or powdered extracts, whenever possible, older children should receive their medication in the form of freshly prepared pills or capsules. Whenever the necessity arises to administer offensive fluid extracts or tinctures, essential oils and the like, it is best to order them in what I may venture to call "home-made liquid capsules." The liquid medicine and the empty gelatin capsules are prescribed separately, and the patient is directed by means of a dropper to prepare each dose of medicine just before taking it. These "home-made liquid capsules" are quite a boon to patients who are averse to taking nauseous mixtures. By means of these capsules you can readily administer also the tincture of iron chloride, which in solution exerts a very destructive effect upon

Pills or capsules for older children.

Care of the teeth.

the teeth; or the different hygroscopic medicinal agents, such as the iodides, bromides, chloral, etc.

Selection of most palatable preparations.

Unfortunately this convenient way of dispensing non-palatable drugs to older children cannot be taken advantage of in prescribing for small children. Hence, an attempt will here be made to devise other means, based chiefly upon the selection of the fittest and most useful preparations, which will enable the physician to render the giving and taking of medicine an act of benevolence rather than an act of cruelty.

For the sake of convenience and in order to avoid repetition the usual classification of drugs in accordance with their therapeutic effects will here be followed.

Digestants.—Most of the so-called appetizers and digestants, such as the pepsin and pancreatin preparations, can be made pleasant in taste by the addition of sugar or in solution with sweet wine or simple elixir.

Of little medicinal value.

Bitter Tonics.—The simple bitters fully deserve their cognomen, since they are certainly very bitter, and simple, insignificant, in their therapeutical effect. The tinctures of gentian, quassia, and calumba owe their medicinal value chiefly to the alcohol they contain. Their use should, therefore, be discouraged, and, if alcohol be indicated, pleasant wines preferred. Of the so-called peculiar bitters, the cinchona preparations are the chief representatives. As their disgusting taste can almost never be disguised, they should never be prescribed for small children, unless intended as an antimalarial. In malarial conditions quinine can best be given by rectum in the manner suggested by me about nine years ago. A half to one dram of quinine sulphate or bisulphate and a few grains of salt are mixed with the white of an egg, and by means of a large glass syringe and wide but short rectal tube injected into the bowels. The white of egg prevents irritation of the intestine, and together with the salt aids in the absorption of the quinine. Older children should be coaxed to take quinine in freshly prepared capsules. The newer "tasteless" quinine preparations are also deserving of trial, and children not averse to bitter medicines can frequently be induced to take quinine in solution with the syrup of yerba santa, or licorice, or in powder form in sweetened chocolate.

The different liquid iron preparations, such as the official wine and the tincture of the chloride, may be rendered palatable by the addition of glycerin, syrup of orange, and water. Powdered iron goes well with sugar and chocolate.

Mineral Acids.—Insufficient attention is being paid to the medicinal properties of mineral acids in the treatment of diseases of infancy and childhood. These acids advantageously replace bitter tonics and act specifically upon the alimentary canal and osseous system. Children like the taste of most of them if well diluted in sweetened water or in combination with raspberry or orange syrup and water.

Instead of bitter tonics.

Alteratives.—Arsenic, iodine, and mercury are the leading remedies of this group. Arsenic is best exhibited as Fowler's solution in plain water. Syrup of iron iodide with simple syrup forms a palatable and very useful hematinic and alterative for children. Sodium and potassium iodide may be prescribed in peppermint or orange-flower water with a little simple syrup, or in compound syrup of sarsaparilla, or elixir of taraxacum. The same holds good for corrosive sublimate. Calomel, the practitioner's panacea, is readily taken by children in powder form with a pinch of sugar.

Cod-liver oil, the almost indispensable tissue builder in all wasting diseases of children, is the stumbling block of the pharmaceutical reformer. Do what you please, cod-liver oil always tastes like cod-liver oil as long as there is any in the mixture. In infants cod-liver oil may be tried by inunction. The majority of children can be "bought" to like the following emulsion:—

℞	Cod-liver oil	4 ounces	120.
	Extract of malt,		
	Syrup of calcium hypophosphite ....āā	1 ounce	30.
	Glycerin,		
	Powdered acacia ....āā	½ ounce	15.
	Cinnamon water ....q. s. ad	8 ounces	240.

Antipyretics and Antirheumatics.—The best antipyretic for children is water, externally and internally. If coal-tar products and the salicylates are indicated they may be administered in powder form triturated with a little sugar to which a minute quantity of essence of peppermint may be added to impart its taste. In prescribing sodium salicylate in solution its nauseating sweet taste may be disguised by a drop or two of the tincture of nux vomica.

Water the best antipyretic.

Hypnotics and Anodynes.—The selection of pleasant hypnotics and anodynes is rather difficult, and perhaps fortunately so, in view of the very deleterious effect they exert upon the delicate infantile organism. However, sometimes they are indispensable, and in minute doses can readily be made palatable. This is particularly the case with the deodorated tincture and the wine of opium, which can be rendered more or less pleasant in taste in a mixture of glycerin and orange-water. The camphorated tincture of opium is a safer preparation for infants and may be prescribed in althea syrup and water. In dispensing the different morphine derivatives, it is advantageous to add a little syrup or powder of acacia to the mixture in order to avoid the formation of a sediment. In excessive irritability of the stomach, the opiates, the bromides, chloral and the newer hypnotics should be administered by rectum, and on rare occasions morphine may also be given hypodermatically.

Minute doses.

Per rectum and hypodermatically.

Antispasmodics.—Belladonna is the principal drug of this group ordinarily employed in diseases of children. The fluid extract tastes fairly well in combination with licorice and water. Spirit of camphor can be made quite palatable in syrup of wild cherry or simple elixir, and the powdered camphor loses part of its miserable taste in chocolate. The emulsion of chloroform and the compound spirit of ether are useful antispasmodics, and fairly palatable in syrup of orange, or almond, and water.

Stimulants.—Nux vomica, strychnine, ammonia, alcohol, strophanthus, caffeine, digitalis, and sparteine, all call for skillful compounding to make them at least acceptable. The extracts and alkaloids should at all times be preferred to tinctures, infusions, or decoctions. Thank heaven! the times have passed when the greatness of the physician stood in direct ratio to the great quantity of medicine he prescribed! As quick circulatory and respiratory stimulants the ammonia preparations, such as aromatic spirit and the anisated solution, are very agreeable and efficient. It is truly sinful to prescribe ammonium chloride instead.

Extracts and alkaloids.

Heart Sedatives.—There are but few occasions when these drugs are of actual benefit to children. Aconite, the standby of the homeopath, may be given in homeopathic doses well diluted in sweetened water. Aconite, like digitalis, is a dangerous drug in the hands of the ignorant. The indication for aconite is sthenic fever, and there are not many children too vigorous while sick. Bitter-almond water in small quantities and well diluted is a useful addition to a palatable cough mixture. The same may be said of compound syrup of squill.

Dangerous remedies.

Emetics.—Although intended to disgust the patient, most emetics are not disgusting in taste. The wine of ipecac, requiring but small doses to produce the desired results, should be preferred to the syrup or infusion. Whenever a quick emetic is indicated, apomorphine may be used hypodermatically, but very cautiously. No special effort need be made to make emetics palatable. It is to be regretted that emetics are dropping into disuse, since many cases of acute gastritis can be arrested in their incipiency by the timely administration of an emetic.

Indication for emesis.

Laxatives, Cathartics, and Purgatives.—Very few of the many members of these groups are being employed in the children's practice. Calomel and aromatic syrup or tincture of rhubarb answer the purpose in most cases. If castor oil is particularly wanted, an emulsion may be made of the following ingredients:—

℞ Castor oil	1 ounce	30.
Oil of peppermint	5 drops	
Sugar	1 dram	4.
Mucilage of acacia and water q. s. ad	2 ounces	60.

Rochelle salts with a little aromatic spirit of ammonia, glycerin, and water form a pleasant mixture. Podophyllin and aloin are best prescribed in suppositories of cacao butter. Finally, it is well worth remembering that an enema of soapsuds often dispenses with drugging.

Soapsuds enema.

Anthelmintics.—Santonine and calomel, the most efficient vermifuges, are readily taken by children either pure or with a little sugar or chocolate. Their effect is greatly enhanced by enemas of soapsuds and turpentine, or a decoction of quassia wood. All teniafuges are very disagreeable in taste and irritating to the stomach. Male fern, the most active teniafuge, may be exhibited as follows:—

℞ Ethereal extract of male fern	3 drams	12.
Emulsion of chloroform	4 drams	15.
Emulsion of almond q. s. ad	2 ounces	60.

Failure to expel the worm is often due to the fact that an oleoresin is used which is prepared from old male fern.

Fresh drug.

Diuretics and Diaphoretics.—Water is the most palatable and, in many diseased conditions, perhaps, most useful diuretic. It should always be thought of before resorting to offensive medicinal combinations. The alkaline diuretics, such as ammonium, potassium, and sodium acetate, as well as potassium

Water as diuretic.

Sodium benzoate.

citrate, the lithium preparations and sodium benzoate, may be rendered palatable in any medicated water with a little syrup. I believe that sodium benzoate is not receiving due recognition as a therapeutic agent. Being an active diuretic, diaphoretic, expectorant, and antirheumatic, it forms, as fully demonstrated by me ten years ago, an ideal remedy for the grip and similar acute affections. The mode of rendering the "hydragogue" diuretics and "special" diaphoretics more or less pleasant in taste has been suggested when speaking of the "heart stimulants and sedatives." I may also add that high intestinal irrigation often advantageously supplants the internal administration of drugs.

Creosote best cough remedy.

Expectorants.—Anisated solution of ammonia, compound syrup of squill, and wine of ipecac, which have already been referred to, are quite palatable and efficient expectorants. To these may be added syrup of senega, tincture of cubeb, compound mixture of glycyrrhiza, syrup of wild cherry, syrup of Tolu, and syrup of althea; the last-named syrups serve also as excellent adjuvants. Creosote, the most valuable remedy in protracted coughs due to pharyngeal, laryngeal, and bronchial catarrh, is fairly palatable in a mixture of glycerin and sherry wine or elixir aurantii.

Astringents.—It will usually be found that bismuth and chalk mixture will do well in most cases where astringents are indicated. The following is a pleasant combination:—

℞ Bismuth subnitrate or subcarbonate	4 drams	15.
Chalk mixture	4 drams	15.
Glycerin	3 drams	12.
Syrup of acacia	2 drams	8.
Peppermint water	q. s. ad 2 ounces	60.

Krameria and tannic acid are best administered in enemas of starch and water. The different newer tannin preparations may be given by mouth with aromatic powder or peppermint sugar.

Lavage.

Gastric Sedatives.—Last in line but primary in importance are the gastric sedatives, since a highly irritated stomach will often reject even the most palatable medicine. Cracked ice, cold or hot water, calomel and sodium bicarbonate, lime, peppermint, or bitter-almond water, small doses of bismuth and cerium oxalate, minute quantities of tincture of iodine well diluted in plain or medicated water—are all useful and more or less pleasant gastric sedatives. In continued vomiting of infants lavage advantageously supplants drugging.

In administering medicines to infants it is often very helpful to divide the regular dose into several smaller doses, giving it, if need be, drop by drop until the whole dose is consumed. In this manner the most irritable stomach will frequently retain the medication where it would otherwise reject it. Before prescribing any nauseous medicine the physician should always bear in mind the grand dictum of Hillel,

Drop by drop doses.

"What is hateful to thee, do not unto thy fellow-man."

Finally, let us remember that a great many drugs can nowadays be administered hypodermatically, a method of medication which is especially advantageous in the treatment of very sick children.

ORGANOTHERAPY.

Organotherapeutics, though still in the experimental stage, is rapidly assuming an enviable position in the field of specific medication. This is true especially of the thyroids, and less so of the suprarenals, pituitary and thymus glands.

Their *modus operandi* upon the human economy—whether by regulation of metabolism, or neutralization of specific poisons—is still shrouded in mystery. It is definitely established, however, that they are all of fundamental importance to the health and growth of the human organism. Furthermore, evidence is gradually accumulating which goes to prove that:—

Regulates metabolism and neutralizes poisons.

1. Absence, atrophy or degeneration of the thyroid gland is followed by cretinism and infantilism;

Cretinism.

2. Absence or disease of the parathyroids gives rise to a state of tetany and disturbance of calcium metabolism[1];

Tetany.

3. The suprarenals exert a powerful influence over the dorso-lumbar sympathetic nerve system and upon the circulation (Addison's disease is generally associated with involvement of the suprarenals);

4. Hypertrophy, and particularly tumors of the hypophysis are productive of gigantism or acromegaly, and, finally,

Gigantism.

5. Hypertrophy of the thymus gland is usually associated with "status lymphaticus."

Status lymphaticus.

From a therapeutic point of view the thyroid gland only has thus far met all expectations. It acts specifically in cretinism and myxedema, and is very serviceable also in obesity and pachyderma-

Myxedema.

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[1] Tetany and calcium deficiency forming conspicuous phenomena also of rickets, there is probably an etiologic relation between this disease and the functional incapacity of the parathyroids.

toses. The gland may be administered fresh (in soup) or dry. The dry preparations are usually given in from ½- to 3-grain doses twice daily, until the desired results have been obtained and in smaller quantities thereafter. Engrafting of the sheep's thyroid in the human body has met with some success. The parathyroids are generally employed (gr. $\frac{1}{10}$ to ¼) as adjuvant or substitute of the thyroid.

Hemorrhages. The suprarenal solutions are used principally locally as hemostatic and astringent, *e.g.*, epistaxis, rhinorrhea of divers origin. Internally, usually hypodermatically (5 min. of a 1:1000 solution); its action resembles that of digitalis.

Infantilism. The pituitary gland is highly recommended (gr. ¼) in infantilism, in hay-fever and asthma (topically as well is internally).

The therapeutic application for the thymus gland is thus far limited to pronounced anemias and marasmus. The results are encouraging.

CHAPTER III.

Congenital Malformations.

Congenital malformations depend upon the following causal factors:—

1. Hereditary disposition (*e.g.*, supernumerary fingers and toes).

2. Antenatal constitutional diseases, especially syphilis and tuberculosis (*e.g.*, hydrocephalus and spina bifida).

3. Traumatism during pregnancy (*e.g.*, multiple fractures and dislocations).

4. Extra- or intra-abdominal pressure through pelvic deformities, tumors, etc. (*e.g.*, talipes).

5. Constriction by amniotic bands (*e.g.*, amputations).

CONGENITAL MALFORMATIONS OF THE HEAD.

MICROCEPHALUS.

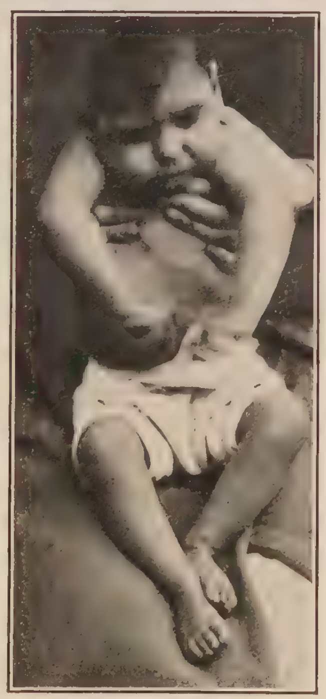

Fig. 29.—Microcephalus—brain disease. (*Sheffield.*)

Brain disease.

Miniature brain.

We distinguish two varieties of microcephalus: One, which appears as a genuine *brain disease* and is the result of antenatal structural disease of the brain (inflammation, sclerosis, cystic degeneration); the other, which presents itself in the form of a *miniature brain* (abnormally small, but not necessarily diseased), is due to congenital arrest of development and leads to premature synostosis of the bone sutures of the skull.

Symptoms of cerebral paralysis.

Microcephalus as a brain *disease* presents typical symptoms of "cerebral paralysis" at birth or soon after. The child suffers from convulsions, rigidity of the entire body, anomalies of sensibility, involvement of the cranial nerves and later disturbances of

Mental backwardness.

locomotion and signs of mental degeneracy, the latter symptoms gradually getting worse. The head is small compared with that of a normal child, but not exceptionally so. On the other hand, in *miniature* brain, the mental deficiency predominates while the physical signs are comparatively slight and are apt to improve as the child grows older (see page 570). (Treatment by means of massage, electricity and baths may improve the paralytic symptoms.)

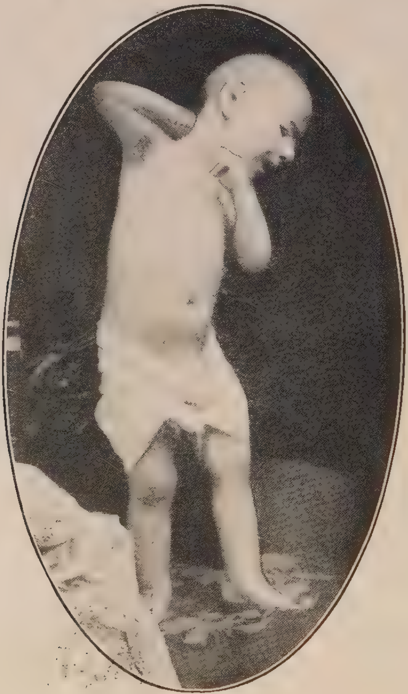

Fig. 30.—Microcephalus—miniature brain. (*Sheffield.*)

CONGENITAL HYDROCEPHALUS.[1]

Large head.

Hydrocephalus is recognized chiefly by the increased size of the head. The enlargement is not always symmetrical on the two sides. All are more or less plagiocephalic, but some are rounded and brachycephalic and others dolichocephalic or scaphocephalic. Hence, the measurements of the head must embrace not only the transverse diameter from one mastoid process across the vertex to the other, and the longitudinal diameter from the glabellum across the vertex to the occipital tuberosity, but particularly the circumference—with the glabellum and occipital tuberosity as centers—the measurement of which greatly exceeds that of the normal child (see page 4). Due allowance, however, should be made for the increased measurement observed in rickety projection of the parietal bones. In the typical hydrocephalic the fontanelles are widely open, the sutures separated and the bones yield to pressure with the finger, usually to a much greater extent than in rachitis. The head sometimes attains an enormous size, so that the child is often unable to hold it up—it shakes to and fro and from side to side—and contrasts strangely with the delicate, emaciated face. The skin of the head is very thin

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[1] See "Acquired Hydrocephalus," page 516.

[illegible]

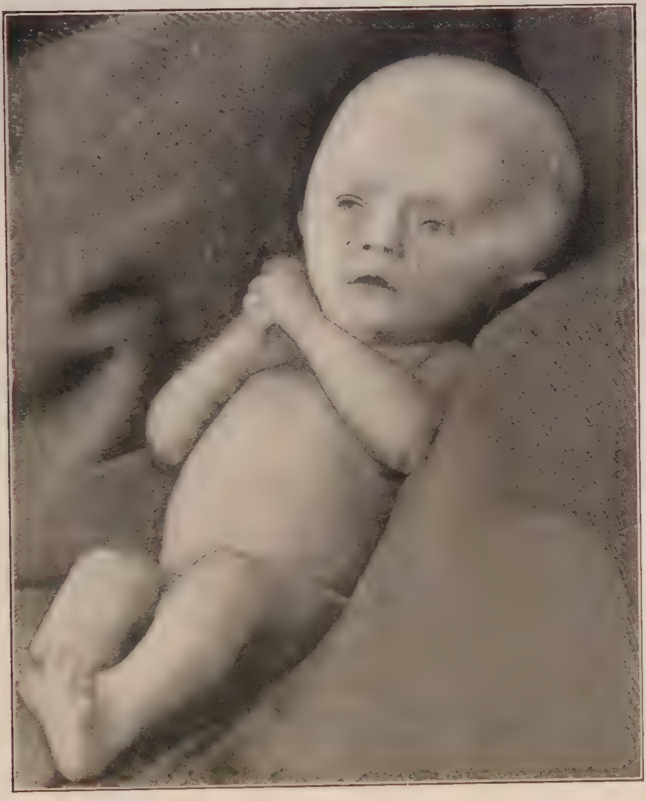

Fig. 31.—Congenital Hydrocephalus. (*Sheffield.*)

Open fontanelles and sutures. Parchment-like consistence of cranial bones.

and tense and traversed by dilated veins. The orbital plates are pushed downward and the eyeballs forward, so that the lids remain partially retracted, leaving a ring of the sclerotic exposed. Hence the peculiarly staring expression, which is greatly exaggerated by the not infrequent accompaniment of strabismus, nystagmus, and optic atrophy—the result of pressure.

The brain symptoms of true hydrocephalus depend upon the amount of cerebrospinal fluid and relative size of the skull cavity and the resultant pressure atrophy of the brain. Where the brain remains unimpaired, the child may grow up apparently healthy in mind and body. This may occur, though less frequently, also with cases in which the disease comes to a standstill. In the majority of instances, however, the symptomatology of hydrocephalus is very definite and progressive in character. Vision and hearing are frequently defective; intelligence is impaired and ranges between simple dullness up to total idiocy. Not rarely hydrocephalus is associated with paraplegia, epileptiform attacks, disturbance of the motor functions and spastic contractures of the upper extremities, spasmus glottidis, and similar spasmodic manifestations. In congenital cases the course of the disease is rapid and death usually occurs in the first few months of life in consequence of marasmus (notwithstanding good appetite and perfect digestion), intercurrent diseases, convulsions and coma. The prognosis is most favorable in syphilitic hydrocephalus, especially if specific treatment is begun early. Antisyphilitic medication should be tried in all cases irrespective of cause, and where the exudation is marked this should be supplemented by lumbar puncture (once a week), or possibly puncture of the lateral ventricles followed by firm but even strapping of the skull. Cases of hydrocephalus with idiocy, spina bifida, etc., are best left alone.

Defective vision, hearing and intelligence.

Paralytic symptoms.

Marasmus.

Spinal and cerebral puncture.

Strapping.

CEPHALOCELE (HERNIA OF THE BRAIN).

Meningocele, Encephalocele, Encephalocystocele or Hydrencephalocele.—Congenital defects in the cranial bones permit the protrusion of a portion of the contents of the skull. The hernia may consist of:—

Differentiation from false meningocele.

(*a*) Meninges (which form the hernial sack) with or without cerebral fluid—*Meningocele.*[1]

(*b*) Meninges and brain substance—*encephalocele.*

(*c*) Meninges and brain substance, which enclose a cavity which is filled with fluid and communicates with a cerebral ventricle—*hydrencephalocele* or *encephalocystocele.*

In accordance with their location we distinguish the following forms of cephalocele:—

(*a*) Cephalocele occipitalis superior—situated above the external occipital protuberance.

(*b*) Cephalocele occipitalis inferior—situated below the protuberance.

(*c*) Cephalocele nasofrontalis—emerges from above the nasal bones.

(*d*) Cephalocele nasoethmoidalis—situated below one of the nasal bones.

(*e*) Cephalocele naso-orbitalis—appears at the inner angle of the eye.

Reducible on pressure.

The presenting tumor varies in size from a small nut to a fetal head. It may be flat, sessile, hemispherical, pear-shaped or pedunculated. Small tumors are soft and elastic, larger ones pulsate and are often translucent. They enlarge during crying, and may be reduced in size by compression,—a procedure which is usually attended by meningeal disturbances. By bearing in mind the characteristic signs, there ought to be no difficulty in differentiating cephaloceles from extracranial cysts, hematomas, abscesses, etc. The diagnosis may be facilitated by an X-ray examination, showing the edges of the opening in the bone. Cephaloceles may remain small and give rise to but very little disturbance. As a rule, however, they grow rapidly and produce death from meningitis, convulsions, or rupture, or proceed a slower course manifested by more or less pronounced backwardness in physical and mental development and other evidences of organic brain disease.

Treatme

Protection.

Small cephaloceles require no surgical interference, but merely protection against external injuries by suitable caps, etc.,

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[1] Congenital meningocele is not to be confounded with acquired so-called pseudomeningocele or meningocele spuria s. traumatica, which is either a result of trauma during delivery or a carious process, especially syphilis. Here the tumor is usually situated at one of the parietal bones, increases in size with the development of the brain or enlargement of the cleft in the bone.

Reposition and compression.

or gentle compression after reposition of the protrusion. Inoperable cases are those complicated by pronounced flattening or diminution in size of the skull, by hydrocephalus or other serious malformations, or where the cleft in the skull reaches down to the foramen magnum. In all other cases removal of the growth is the only proper treatment, followed, if necessary, by osteoplastic closure of the defect in the skull.

Early operation.

The operation is not rarely successful, if performed by a skillful surgeon, and, in view of the extremely grave prognosis in large tumors if left alone, there is sufficient justification for early (!) surgical interference.

CONGENITAL MALFORMATIONS OF THE FACE.

Including those of the Palate, Mouth, Eyes, Nose, and Ears.

CLEFTS OF THE FACE AND LIPS.

1. **Median,** the result of non-union of both globular processes of the central nasal process. This cleft is rarely extensive.
2. **Lateral** (Labium Leporinum, Harelip, Cheiloschisis), produced by failure of union of one or both globular processes with the superior maxillary processes. Clefts of the upper lip may accordingly be unilateral or bilateral, may exist as a mere notch into the skin margin of the lip, or more frequently extend for some distance upward, involving the whole lip, nostril and upper jaw. It is occasionally associated with cleft palate.
3. **Oblique** (Meloschisis), arises from defective closure of the groove between the lateral nasal process and the superior maxillary process. The cleft runs as high as the lower lid.
4. **Transverse** (Macrostoma), as a result of patency of the groove between the superior maxillary process and the first branchial arch (mandibula).

Occasionally fistules and fissures are observed in the bridge of the nose and lower lip.

Surgical treatment.

For details of treatment the reader is referred to text-books on Surgery.

CLEFT PALATE (PALATUM FISSUM, PALATOSCHISIS).

It is due to defective union of the processes of the superior maxillary and palate bones which during intra-uterine life normally grow inward to meet the vomer in the middle line and the intramaxillary bone in front to form the hard and soft palate.

1. **Complete** (Uranoschisma).—The fissure extends in the middle line through the uvula and the soft and hard palate, and thence through the alveolar process in the line of suture either on one or both sides of the intramaxillary bone. It is generally combined with double or single harelip, and is then designated "Wolf's Jaw."

Wolf's jaw.

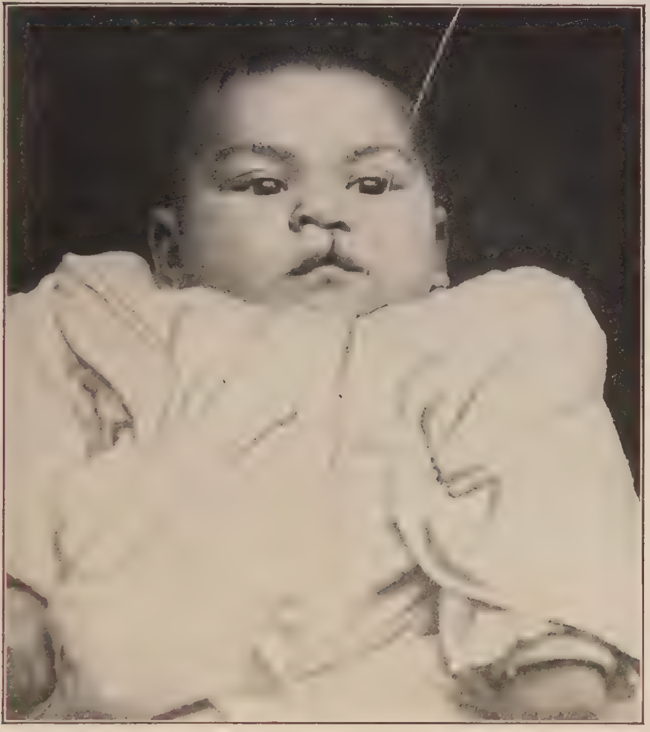

Fig. 32.—Hare-lip. (*Sheffield.*)

2. **Partial** (Uranocoloboma).—It may involve the uvula only, or part of the soft and hard palate as well. Sometimes it is limited to mere notching of the alveolar process on one or both sides and forms the continuation of uni- or bi-lateral harelip.

Difficult feeding.

The consequences of cleft palate, if extensive in degree, are by far more serious than those of cleft lip. Suction and deglutition are greatly interfered with. In older children the voice, articulation, sense of taste, smell, and hearing may all be impaired.

The management of cleft palate is principally surgical. The

Early operation.

earlier the operation is undertaken the more perfect are the results. The mode of feeding frequently presents great difficulty. Infants born with marked cleft palate who are unable to nurse have to be fed artificially either with the spoon or through a tube passed through the nose into the stomach. A rubber plate covering the defect in the palate often acts admirably

DEFECTS OF THE MOUTH AND TONGUE.

Atresia Oris (Microstoma).—The lips may be grown together partially or completely. In the latter event an immediate plastic operation is inevitable. Congenital microstoma should not be confounded with the acquired contractures of the oral orifice resulting from syphilis, gangrene, burns, etc.

Adhæsio Linguæ (Ankyloglossia, Tongue-tie).—It is produced by a large and anteriorly displaced frenulum, and varies greatly in degree, the insertion of the frenulum sometimes extending so far forward as to interfere with suckling and, later, with speech.

Dangerous hemorrhage.

The anomaly may be removed by nicking the frenulum with a scissors, and further "loosening of the tongue-string" with the finger, thus avoiding injury to the ranine artery (dangerous in hemophilia). The rare adhesion between the epithelial surfaces of the tongue and floor of the mouth can be liberated in a similar manner.

Differentiations from cretinism.

Macroglossia (Large Tongue).—Enlargement of the tongue may be due to a true lymphangiomatous tumor (cavernous macroglossia), or to a fibrous hypertrophy (fibrous macroglossia). Both forms may coexist. The tongue may be so markedly enlarged as to find no room in the mouth, and by protruding from it become bruised, chapped and cracked, assume such dimensions as to render suckling very difficult or impossible, and possibly lead to a fatal issue from inanition. Congenital macroglossia from the aforementioned causes is not to be mistaken for protrusion of the tongue associated with cretinism. Mild degrees of macroglossia usually improve spontaneously, with the growth of the oral cavity; severe forms call for removal of a wedge-shaped piece of the protruding tongue.

MALFORMATIONS OF THE EYES.

Anophthalmus (Absence of One or Both Eyes).—This is a rare malformation. ~~In a great~~ many cases careful anatomic examination reveals the presence of rudimentary eyes. If only one eye is absent the existing eye may be perfectly normal or defective in various ways.

Microphthalmus.—An adbnormally small eye causes more or less severe disturbance of vision which may in some instances be relieved by suitable glasses. It is sometimes associated with adhesion of the edges of the eyelids (*ankyloblepharon, cryptophthalmus*), and other abnormalities of the bulb, which may require surgical treatment.

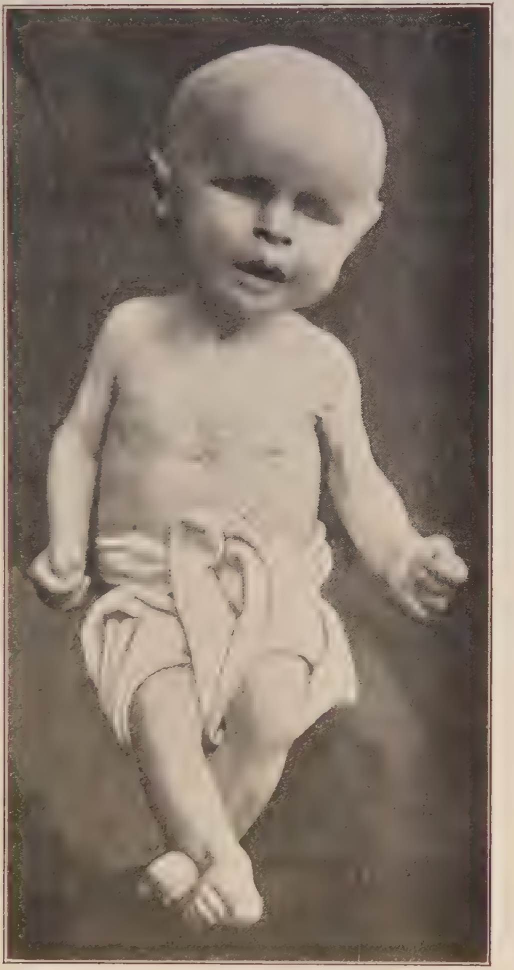

Fig. 33.—Bilateral Anophthalmia. (*Sheffield.*)

Eyeglasses.

Operative treatment.

Differentiation from cataract.

Atresia Pupillæ Congenita.—Occasionally the pupillary membrane persists after birth and varying with its extent leads to more or less grave defective vision. The fine, gray membrane may be mistaken for an exudation or capsular cataract. Spontaneous improvement is the rule.

Cataracta Congenita.—It is usually partial, rarely complete. It may exist in the form of limited opacities and not be recognized until school age. In the complete variety the condition may present a white pupil.

Coloboma Iridis (Iridoschisma, Fissure of the Iris).—It is usually bilateral and sometimes associated with coloboma of the choroid, fissure of the upper eyelids without involvement of the external skin, microphthalmus, and cataract. If uncomplicated, it disturbs vision but slightly.

Albinism.

Irideremia (Aniridia).—Partial or complete absence of the iris usually occurs on both sides and is associated with abnormality of the cornea and poor vision. The pupils are iridescent like cats' eyes, and owing to too strong perception of light, the affected children convulsively open and close the eyelids. The same phenomenon is often observed in *albinism*—a condition in which there is a congenital deficiency of pigment in the iris and choroid. Albinos have a blue iris and very fair complexion.

Exclusion of light.

✓ Exclusion of superabundance of light by means of dark glasses or artificial diaphragm.

MALFORMATIONS OF THE NOSE.

Adhesions between the turbinated bones, particularly the inferior, and the septum. The adhesions may be membranous or bony, and not rarely associated with deflection of the septum. The treatment is the same as in the acquired conditions. Surgical-

Difficult suckling.

Atresia of the Posterior Nares.—The closure may be membranous or bony; in the latter condition there is bony union between the palate and the sphenoid. If the closure is only moderately firm, it can be perforated by a stout probe or galvanocautery. Firm bony union giving rise to difficult suckling calls for the employment of chisel and mallet or trephine, using the finger in the nasopharynx as a guide to prevent the instrument from penetrating too deeply.

MALFORMATIONS OF THE EAR.

Fissures and Fistulas of the Ear.—*Fissures* (beneath the tail of the helix) and *fistulæ* (in front and above the tragus) are occasionally observed, especially in connection with other congenital malformations. Deep fistulæ sometimes secrete a serous fluid, not rarely causing intractable eczema and requiring operative interference.

AURICULAR APPENDAGES in the form of scattered round or oblong, smooth or warty pieces of cartilage are not rarely found in front of the ear. They can readily be removed by knife or cautery.

EAR PROMINENCE is a malformation which can often be remedied in the newly born by keeping the ear properly bandaged for several weeks. Sometimes it calls for a slight operation.

Atresia auris, absence of the auditory meatus, is most frequently complete, involving the cartilaginous as well as the bony portion of the canal. Moreover there is usually also an abnormal tympanic cavity. Hence very little benefit can be expected from operative interference.

Often abnormal tympanum.

All sorts of ear deformities are encountered in connection with idiocy and the allied mental deficiencies (*q. v.*).

MALFORMATIONS OF THE LARYNX AND TRACHEA.

Congenital Diaphragm of the Larynx.—The glottis is more or less occluded by a membrane running transversely across the vocal cords. The symptoms stand in direct relation to the size of the remaining opening.

In marked cases the membrane should be excised after preliminary tracheotomy.

Laryngocele and Tracheocele (Aerocele).—The tumor is situated laterally or in the median line. It increases in size on coughing or crying and diminishes on pressure.

Enlarges on crying or coughing.

The treatment consists of excision of the cyst and closure of the communication with the respiratory tube.

Stridor Congenitus (Child-crowing).—This congenital anomaly is not to be confounded with laryngospasmus (spasmus glottidis, see page 562), which is an acquired affection and forms a symptom of spasmophilia (*q. v.*).

The etiology is still indefinite, though in a number of cases the stridor could be traced to malformation of the epiglottis and hypertrophy of the thymus gland.

Stridor congenitus is manifested by a loud, crowing inspiration, accompanied by retraction of the jugulum and epigastrium. It is free from cyanosis or any systemic disturbance, and usually subsides spontaneously in the course of a year or so.

Free from cyanosis.

MALFORMATIONS OF THE NECK.

Fistula Colli Congenita.—It is a rare anomaly, the result of defective closure of the second and third branchial arches. The fistula is situated either laterally immediately above the sternoclavicular articulation or medially at a varying level between the hyoid bone and the jugulum. The fistula becomes apparent by its fine, pinhead-sized opening with an irregular, moist surface. By passing a fine probe the fistula is found to end either blindly or in the pharynx or esophagus. As long as its track is free, the fistula gives rise to no serious symptoms. Its occlusion, however, is associated with danger of retention of the mucoid secretion and cyst formation. (Hence the indication for complete extirpation of the fistulous canal.)

Branchial Appendages.—They occur in the shape of warts, nipples or mushrooms, along the margin of the sternomastoid, between the sternoclavicular region and the hyoid bone, consist of skin alone or of skin and cartilage, and are frequently associated with auricular attachments (*q.v.*). They cause no annoyance except from a cosmetic point of view. They are readliy removable and non-recurrent.

[illegible] to your wrist

Branchiogenetic Cysts.—The seat of these variously sized (from a small nut to a hen's egg), elastic, serous, seromucous, sebaceous, sometimes dermoid cysts is the anterior region of the neck (in the middle line or at the side). The cyst contents may become purulent through infection or sanguinolent through involvement of a blood-vessel. Aspiration is a useful aid in the diagnosis, and extirpation of the cyst the only rational mode of treatment.

Danger of infection.

Hygroma Cysticum Colli Congenitum (Lymphangioma Cysticum).—This tumor consists of a number of small or large communicating or non-communicating cysts. It varies in size from a slight swelling under the lower jaw or over the clavicle to an enormous tumor embracing the whole neck, and extending downward to the chest and upward to the face. It may even involve the mouth, throat, base of the cranium and mediastinum. In the latter event the prognosis is extremely grave. As the removal of large tumors is attended by great difficulties, it is often justifiable first to try aspiration with subsequent injection of iodine or incision and antiseptic packing. Small hygromas should unhesitatingly be extirpated.

Cervical Rib.—The supernumerary rib is a hard, bony clasp which begins at the seventh cervical vertebra and either ends there as a small protuberance or continues farther to join the first thoracic rib, or even the sternum. The symptoms depend upon the degree of pressure exerted by the cervical rib upon the neighboring structures, especially the subclavian artery and some branches of the brachial plexus.

Pressure paralysis.

Cervical rib may be confounded with exostosis of the first rib, tumor (also tuberculous glands) in the supraclavicular fossa, or cervical spondylitis. Exostosis and spondylitis are best diagnosed by means of a careful X-ray examination. A tumor is softer and movable.

Differentiation from exostosis or spondylitis.

In the event of marked disturbances the supernumerary rib should be resected, care being taken not to injure the pleura.

Resection.

MALFORMATIONS OF THE THORAX.

Defects of Sternum.—Partial or complete absence or smaller congenital clefts of the sternum are of rare occurrence. They give rise to hernial protrusions of the lung which if small in size are apt to be mistaken for soft tumors or abscesses. Lung hernia is reducible on pressure, changes in size and shape with respiration and is frequently associated with paroxysms of coughing.

Protrusion reducible on pressure.

Among the divers deformities of the sternum, congenital, non-rachitic "funnel chest" is deserving of special mention. It differs from congenital or acquired rachitic funnel-shaped chest by the absence of other rachitic deformities.

Anomalies of the Ribs.—One or more ribs may be absent or rudimentarily developed. (The intervening space is filled with membrane.) There may also be accessory ribs (see Cervical Rib), or several ribs may be united.

Defects of the Thoracic Muscles.—Congenital, partial or total absence of one or several of the thoracic muscles is apt to

Resembles muscular dystrophy.

be mistaken for progressive muscular dystrophy. The former, however, is unilateral, while the latter is bilateral. Secondary scoliosis is apt to follow the congenital muscular defects.

All the aforementioned malformations of the thorax require some mechanical contrivance, to prevent either injury to the internal structures or secondary deformities.

MALFORMATIONS OF THE ALIMENTARY TRACT.

Regurgitation of food.

Atresia Œsophagi.—Congenital esophageal strictures are very rare. They give rise to difficulty of swallowing and immediate regurgitation of the food through the mouth and nose.

The treatment is the same as in acquired esophageal strictures. Owing to the absence of true scar tissue in the congenital form, the prospects of recovery are brighter.

STENOSIS PYLORI CONGENITA.

Stenosis of the pylorus may be complete or partial. *Complete atresia* is extremely rare and invariably fatal within a few days after birth—before the diagnosis can be established.

Partial stenosis of the pylorus, on the other hand, is a comparatively frequent affection which not rarely terminates in recovery, either spontaneously or through medical and surgical treatment. It is distinguishable in two forms: True and False.

Organic narrowing.

1. *True* or hypertrophic pyloric stenosis is invariably due to a congenital narrowing of the lumen of the pylorus and is associated with more or less primary hypertrophy of the pyloric ring.

Spasmodic contraction.

2. *False* or spastic pyloric stenosis (pylorospasm) is the result of congenital faulty innervation of the stomach, or of acquired digestive and nervous disturbances. It is free from primary hypertrophy of the pyloric ring. Sooner or later secondary hypertrophy of the muscular and mucous coats of the stomach occurs in consequence of the increased force required by the stomach to propel the ingesta. At a later stage of the disease the stomach walls lose their tonicity and dilatation is a frequent complication.

The diagnosis of true ~~pyloric~~ stenosis usually presents no difficulty, and it can readily be distinguished from atresia of the esophagus or duodenum by bearing in mind the typical clinical picture of the disease. The apparently fully developed infant at birth, after a period of well-being of from a half to three weeks, begins to vomit sometime after each feeding. The vomiting rapidly becomes very violent in character, and the contents of the stomach, which appear greater (*ischochymia*—retention of digested food) than the child could have taken in one feeding and consists of a hyperacid[1] mixture of mucus, digested and undigested food, free from bile, is explosively ejected. As an immediate result of the vomiting, the intestinal tract remains empty; hence, pseudoconstipation or only occasional evacuation of a small quantity of brown, foul-smelling fluid. The urine is scanty and concentrated. The infant acts very hungry, voraciously swallows a few mouthfuls of food, but, being seized by sudden spasmodic pain, drops bottle or breast, only to grasp it again after some relief prevails. The abdomen is sunken in, while the epigastrium is distended, and here and there are visible peristaltic movements (*hyperkinesis*) of the stomach, from right to left. In some cases a tumor—the hypertrophied pylorus—is palpable a little to the right of the stomach and in cases of long standing there is usually more or less marked gastric dilatation.

Excessive vomiting; remnants of previous feedings.

Absence of bowel movements.

Visible peristalsis.

In *pylorospasm* the symptoms are much less pronounced, but otherwise cannot be distinguished from true pyloric stenosis. The course of the affection varies with the degree of the contracture. In the majority of instances the *true* form of the disease, if not operated upon early, terminates fatally in from four weeks to four months, with symptoms of inanition and collapse. Occasionally, however, a change for the better occurs and slow recovery follows. This is particularly apt to take place in spastic pyloric stenosis, especially if early and properly treated. With these facts in view, it is extremely difficult to decide when, and whether, surgical intervention is indicated. The profession is greatly divided on this question. The statistics adduced for and against an operation seem to favor both contentions. The surgical "cures" do not always assure us of their permanency.[2] On the other hand, who can vouch for the permanency (remissions are not rare!) of the medicinal "cures," and for the correctness of the diagnosis in such cases! Appreciating, then, the gravity of the prognosis of true pyloric stenosis even under the best medical management, and the recent grand achievment in stomach

Prognosis bad, with or without operation.

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[1] In one case under our observation there was total achylia gastrica.

absence of ferm
of gastric ju

[2] A little patient of mine, nine weeks old, recently operated upon, did well for six days, but died two days later from the effects of a minute gastrointestinal fistula.

surgery, it is justifiable *after two weeks' faithful but unsuccessful trial of dietetic and medicinal measures* to recommend an operation, namely:—

1. In *bottle*-fed infants presenting the usual symptoms of pyloric stenosis, *plus pylorus-tumor.*

2. In *breast*-fed infants presenting the usual symptoms of pyloric stenosis, even *minus* palpable pylorus-tumor.

Early operation in true stenosis.

An operation, if indicated, should not be delayed until the child is at death's door. The choice between divulsion (Loreta's), pyloroplasty and gastroenterostomy depends upon the pathological condition of each individual case.

Small feedings.

The *non*-surgical treatment of congenital pyloric stenosis must be carried out systematically and faithfully. Whenever possible, the infant should be fed on woman's milk, preferably with a spoon or tube, in order to gauge the amount of food consumed and possibly retained by the infant. The amount of each feeding should not exceed one ounce, but may be given every hour, so as to sustain the child's vitality. Modified or predigested milk may be administered instead of woman's milk if the latter is not readily obtainable. In view of the fact that almost two-thirds of the cases of pyloric stenosis thus far reported were breast-fed babies, one is tempted to recommend cows' milk feeding as a therapeutic or, at least, prophylactic measure against pyloric stenosis. Indeed, following the temptation in one of my own cases I was—perhaps accidentally—rewarded by happy results. May I venture to suggest that the large curd of cows' milk tends mechanically to dilate the contracted pyloric orifice?

Lavage.

Hot poultices.

Reduction in the frequency of the attacks of vomiting and in the amount ejected forms the first and best indication of improvement in the condition. Next to careful feeding, systematic washing of the child's stomach serves as the sheet-anchor in the therapeusis of congenital pyloric stenosis. It should be practiced at least twice a day with plain, cool (60° to 70° F.) water, occasionally adding a small amount of bicarbonate of soda to neutralize the hyperacidity of the stomach. The washing should be continued until the water returns clear. The effects of the lavage are the removal of decomposing substances from the stomach, arrest of fermentation and allayment of pain and spasm. For the latter purposes prolonged warm baths and hot compresses to the epigastric region are also very useful. To counteract the excessive loss of fluids, a daily enteroclysis or hypodermoclysis is of advantage. Internal medication is of little value except anodynes for the relief of pain and spasm. For this purpose minute doses of codeine with or without belladonna may be administered in the form of suppositories.

Anodynes.

Skillful nursing privately or in hospital should be insisted upon.

CONGENITAL STENOSES AND ATRESIÆ OF THE INTESTINES.

Any portion of the intestines may be congenitally malformed or completely obliterated. Partial stenosis is most frequently observed in the small intestine, while complete atresia in the rectum and anus. As in acquired intestinal obstruction, the lumen of the intestine above the occlusion is widely dilated, while that below it is more or less collapsed.

The symptoms vary with the seat of the lesion. The higher the stenosis, the earlier and more pronounced the vomiting, the larger the quantity of the meconium, and the more marked the dyspnea and eventually the cyanosis as a result of compression of the thoracic organs by the highly distended stomach.

Fecal vomiting, meteorism and anuria.

On the other hand, the lower the stenosis, the more fecal the vomiting, the greater the meteorism, and the more marked the disturbances of the bladder and kidney (partial or total anuria as a result of compression of the ureters by the highly distended intestines). In stenosis of the duodenum the vomitus contains bile substances.

Complete stenosis fatal.

Associated with the local symptoms of intestinal stenosis are: dry tongue, subnormal temperature, rapid emaciation, pinched features of the face, and collapse. Death usually takes place within a week. Where the stenosis is only partial and slight, the child may linger for months and ultimately recover.

Operation.

In mild cases the treatment should be symptomatic, principally to relieve constipation and to mitigate the pain and agony. Surgical intervention as a last resort.

CONGENITAL HYPERTROPHY AND DILATATION OF THE COLON.

(Megacolon Congenitum, Hirschsprung's Disease).

This congenital affection should not be mistaken for acquired dilatation of the large bowel associated with intestinal atony from various causes.

Greatly distended abdomen.

Constipation alternating with diarrhea.

The congenital dilatation is manifested soon after birth by retention of the meconium, although the child is otherwise apparently healthy and free from congenital stenosis of the anus or rectum. Intestinal irrigation brings forth but a small quantity of feces. The infant is restless and constipated, and its abdomen gradually becomes greatly distended. Some time later the constipation is followed by more or less copious diarrhea due to irritation from retained feces. After expulsion of the stool and gases the abdomen is reduced in size, but after a short time it again becomes distended, giving rise to the aforementioned symptoms. Most infants succumb early to the disease, from interference with the thoracic organs or autointoxication by the decomposing intestinal contents; others may live longer and in rare instances even entirely recover.

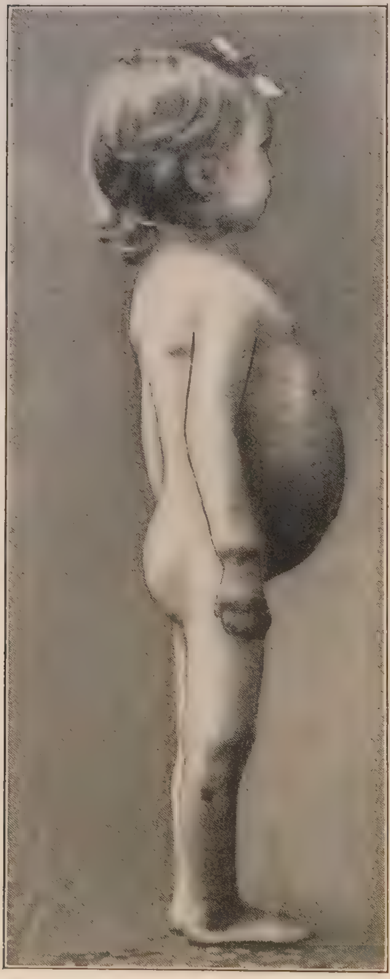

Fig. 34.—Megacolon Congenitum (3 years old). The size of the abdomen is considerably reduced after high enema. (*Sheffield.*)

Pathologic findings.

Post-mortem examination reveals either of the following conditions:—

1. Simple dilatation and often lengthening of the colon; 2, ectasis of a section of the colon with or without compensating dilatation or hypertrophy of the adjoining portions; 3, general enlargement of the intestinal lumen and hypertrophy of its walls. The hypertrophy usually involves the longitudinal and circular muscular fibers.

The treatment is chiefly symptomatic (see Constipation). In severe cases surgical intervention.

Symptomatic treatment, or eventually operation.

ATRESIA OF THE RECTUM AND ANUS.

(*a*) **Atresia Ani Proper (Imperforate Anus).**—The rectum is normal and ends blindly into the completely closed anus.

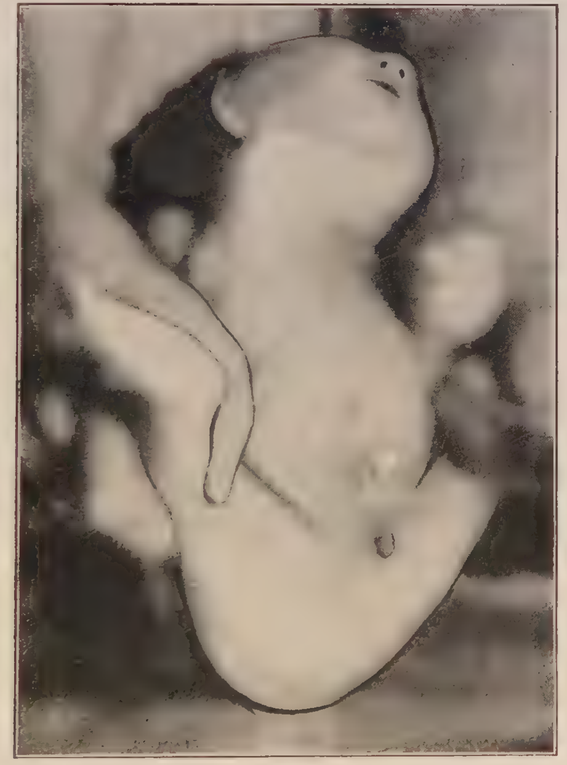

Fig. 35.—Congenital Absence of Scrotum and its Contents, Anus and Rectum. (*Sheffield.*)

There may not be the slightest indication of an anus, or the latter is indicated by a few comb-like prominences, a small fossa, or a round induration.

(*b*) **Atresia Recti.**—The anus is normally developed, but the rectum ends blindly somewhere higher up in the canal.

(*c*) **Atresia Ani et Intestini Recti.**—In this condition the anal orifice is absent and the rectum is arrested in its development higher up, usually in the region of the sacroiliac symphysis.

(*d*) **Atresia Ani Complicata.**—There is atresia of the anus, and the rectum terminates either (1) in the bladder (atresia recti vesicalis); (2) in the vagina (atresia recti vaginalis), or somewhere in the urethra (atresia recti urethralis).

(*e*) **Atresia Recti cum Fistula.**—The anus proper is occluded; the rectum ends blindly, but is connected with the outer skin by a fistulous tract. The anal orifice is thus located in an abnormal position in the perineum, vulva, scrotum, etc.

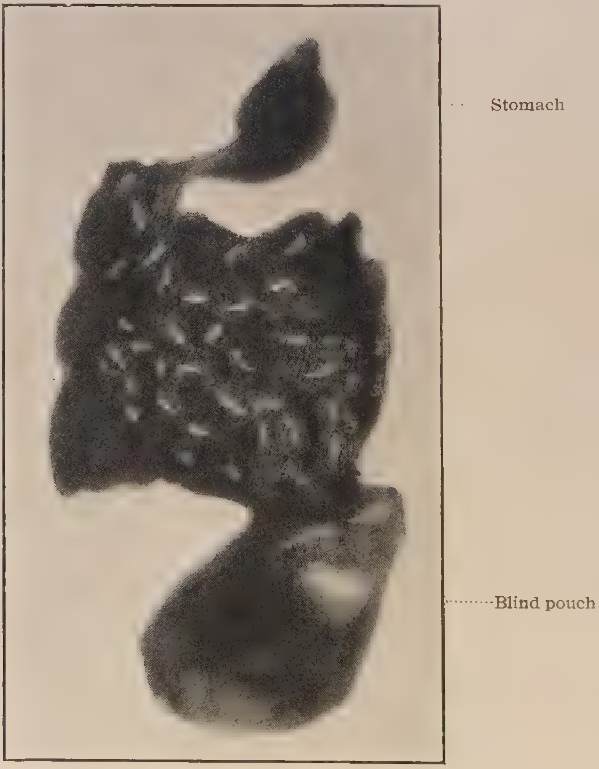

Fig. 36.—Stomach and Intestines of case Fig. 35, showing ending of colon in a blind pouch filled with meconium. (*Sheffield.*)

The diagnosis of imperforate anus or rectum usually presents no difficulty.) Imperforate anus can readily be made out by inspection. Absence of meconium in the presence of a normal anus indicates that the defect is somewhere higher up. Digital or instrumental examination rarely fails to locate the seat of

Absence of meconium.

obstruction. Atresia ani complicata may be detected by the presence of meconium in the urine or by continuous escape of feces from the abnormal communications. The latter symptom is indicative also of the last form (*e*) of atresia, which can readily be seen.

Meconium in the urine.

Imperforate anus and imperforate rectum are the only two conditions giving rise to immediate more or less grave symptoms. The child passes no meconium, appears restless, strains, cries, its abdomen is distended, it suffers from dyspnea, and vomits occasionally. If not relieved it succumbs within a week from rupture of the intestines and peritonitis. Prompt operative interference is therefore imperative. If the obstruction is in the anus, or in the lower part of the rectum, puncture or incision with consecutive dilatation will often suffice to effect a cure. Whenever the point of the atresia cannot be discerned, an artificial anus should be made for quick relief, postponing the curative measures for later. An operation should be postponed also in all other forms of atresia ani or recti, where the escape of meconium is not entirely interfered with.

Symptoms of obstruction.

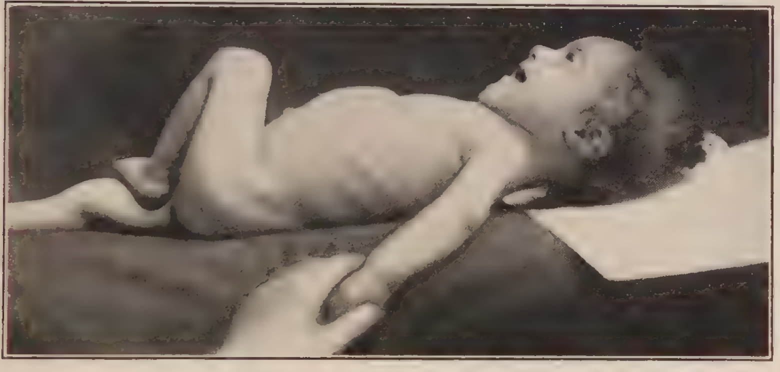

Fig. 37.—Diastasis Recti Abdominis Patient suffering also from amaurotic family idiocy. (*Sheffield.*)

Operative treatment.

DEFECTS OF THE ABDOMINAL PARIETES.

Diastasis Recti Abdominis.—Lozenge-shaped separation of the abdominal wall extending from the xiphoid to the umbilicus is congenital in nature and due to defective closure of the deep layers of the abdominal coverings. It is sometimes associated with umbilical hernia.

Sudden colic.

The symptoms make their appearance when the child is able to run about and jump, and consist of sudden attacks of colic (not to be mistaken for enteralgia!), uneasiness in the epigastric region, pallor, etc., which subside when the child is perfectly at rest. These paroxysms are due to partial incarceration of the stomach in the abdominal slit, and should be remedied by bringing and keeping the separated recti muscles together by means of plaster straps or suitable bandage.

Abdominal supporter.

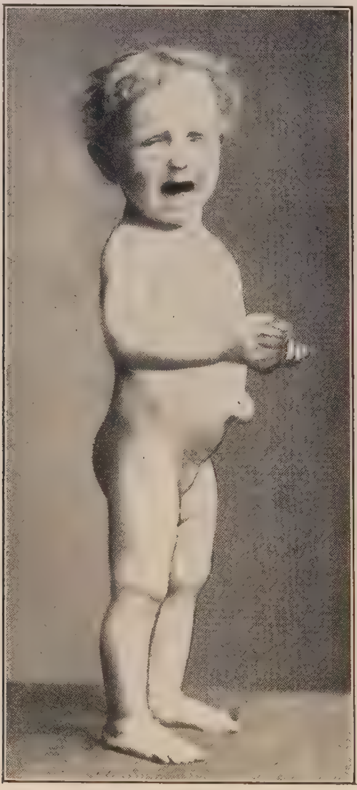

Fig. 38.—Umbilical Hernia. (*Sheffield.*)

CONGENITAL UMBILICAL HERNIA.

(Hernia Funiculi Umbilicalis, Exomphalos, Omphalocele Congenita, Ectopia Viscerum, Amnion Navel).

As a result of faulty development of the abdominal coverings, instead of an umbilicus, a variously sized, saclike dilatation is occasionally observed which may contain intestinal loops, the stomach, liver, spleen, etc. The hernial sac is composed of the amnion and parietal peritoneum. At birth the contents of the sac can usually be recognized through the thin, transparent membranes, but small protrusions into the cord are apt to be overlooked, and carelessly tied off with the umbilical rest. If there is considerable eventration, the infants die early from rupture of the sac and peritonitis. The first indication therefore is to replace the prolapsed structures into the abdominal cavity and to keep them there by means of a suitable bandage.

Small protrusions may be overlooked

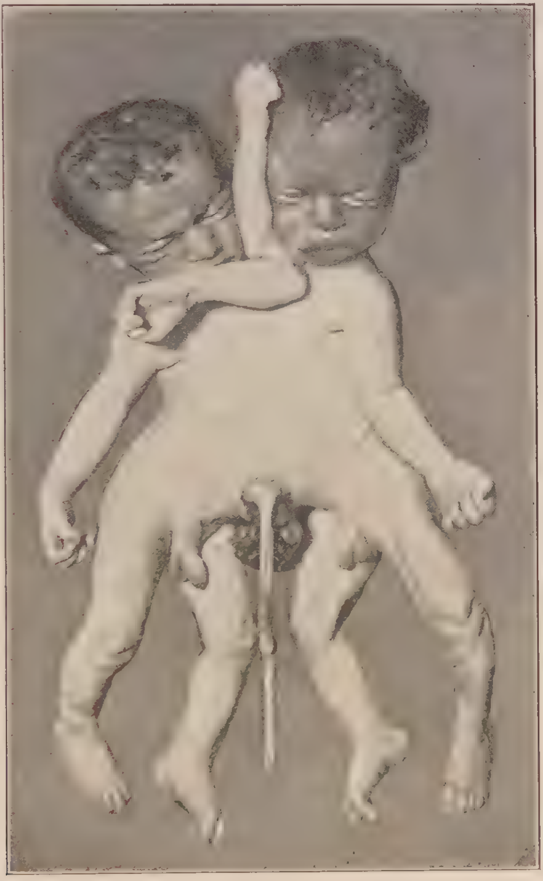

Fig. 39.—Thoracoabdominopagus, with Ectopia Viscerum. (*Sheffield.*)

Reposition of prolapsed portion, and strapping.

Radical operation. In this manner small hernias not rarely subside spontaneously. Large hernias should be treated by a radical operation.

PERSISTENCE OF THE DUCTUS OMPHALOMESENTERICUS.

(Vitellointestinal Duct).

Physiologically, the omphaloentericus duct, the embryonic tubular communication between the intestinal canal and the germinal vesicle, disappears at about the eighth week of fetal life. Occasionally the duct is not obliterated, and leads to the following principal abnormalities:—

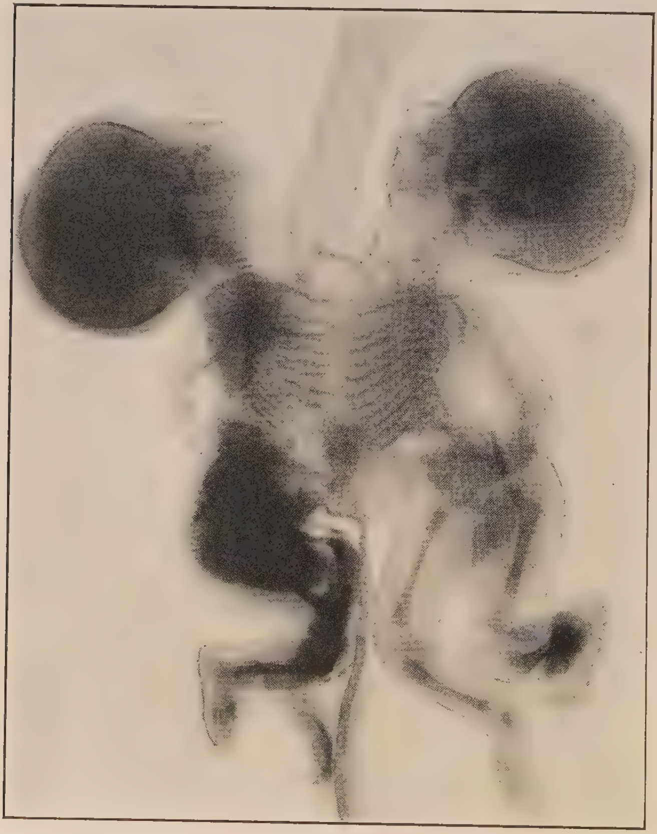

Fig. 40.—Skiagram of Thoracoabdominopagus (same as Fig. 39), with Ectopia Viscerum. (*Sheffield.*)

1. A fine fistula at the umbilical ring, forming a communica-

tion between the bowels and the exterior, and secreting a cloudy fluid containing a trace of fecal matter.

2. A hernial protrusion through the umbilicus in the form of a red finger-shaped tumor which is usually composed of the prolapsed walls of the fistula, but sometimes is composed of intestinal loops.

Danger of intestinal strangulation.

3. Open Meckel's diverticulum. It is a blind appendage of the lower part of the ileum, and may be free or united with the umbilicus by a solid cord. Under certain conditions it may enter a hernial sac and here become strangulated. It may produce "ileus" by incarcerating some loops of the intestines, and give rise to local intestinal inflammation closely resembling that of appendicitis.

Differentiation from persistent urachus, and sarcomphalos.

Persistent omphaloenteric duct may be mistaken for:—

1. Persistent urachus. On examination with the catheter it can be reached through the bladder; the secretion is composed chiefly of urine.

2. Sarcomphalos—has no fistular opening.

Fine fistulæ frequently close after repeated cauterization with the caustic stick. Wherever the prolapse is very marked or in cases associated with open diverticula a radical operation is imperative, since their presence is always a menace to life.

URACHUS FISTULA.

(Fissura Vesicæ Umbilicalis).

Escape of urine through umbilicus.

Persistent urachus—the duct through which the urinary bladder communicates with the allantois—gives rise to a fistulous tract which ends at the umbilicus. On pressure a small hernial tumor arches forward and secretes a clear or turbid fluid, composed of urine alone, or urine, mucus and pus. If the fistula is large, the flow may be continuous. It may give rise to cystitis and even pyelonephritis compelling early operative procedures. The first attempt at a cure should be directed to making the natural outlet quite free (*e.g.,* cure of phimosis). Small fistulæ often yield to cauterization and continued pressure with a bandage. If this fails, the walls of the sinus should be freshened and then sutured.

Its differentiation from persistent ductus omphalomesentericus has been emphasized above.

MALFORMATIONS OF THE GENITO-URINARY ORGANS.

CONGENITAL ABNORMALITIES OF THE KIDNEYS.

Deformity.

Absence.

Displacement.

The kidneys, like all other parts of the body, are subject to defective embryonic development. They may be abnormal in size, shape (horseshoe) and number. This is of clinical importance, since malformed kidneys are more easily affected by disease, especially tuberculosis, than normal organs. *Congenital absence* of one kidney has been observed once in about 4000 autopsies. Furthermore, it is usually found that, whenever one kidney is absent, the other one is in a more or less diseased condition, chiefly greatly hypertrophied. *Congenital displacement* of the kidney (both kidneys on one side; in front of the vertebral column; low down in the pelvis) is very apt to cause many diagnostic errors.

MALFORMATIONS OF THE URETERS.

Secondary involvement of kidneys.

Abnormal ureteral openings, as to size and position, are of great clinical significance. In the male the ureter may terminate into the sphincter of the bladder, the prostatic portion of the urethra, or in the seminal vesicle, and by interference with the flow of urine give rise to dilatation of the ureter and renal pelvis and atrophy of the renal parenchyma. In the female the ureter may end in the sphincter of the bladder, in the urethra, or in the vagina. More serious than misplacement is *absence* or *atresia* of the ureter. Either one of these latter conditions invariably produces *hydronephrosis,* compelling extirpation of the affected kidney. *Double ureter,* if free from any other anomaly, is not attended by any pathologic phenomena.

MALFORMATIONS OF THE BLADDER.

Round, moist red mass at hypogastrium.

Ectopia Vesicæ Congenita, Cleft Bladder, Fissure of the Bladder, Exstrophy Vesicæ.—Cleft bladder arises from arrest of development of the anterior walls of the bladder and abdomen, and often also of the symphysis. It may be partial or complete. In the complete variety the posterior vesical wall protrudes as a round, moist, bright-red tumor, through a gap in the abdominal wall, situated in the median line between the umbilicus and the urethra. The mass is marked by two small tubercles on both sides—the orifices of the urethra—

from which the urine dribbles continuously. In the male this is associated with epispadias of the rudimentary penis; in the female the clitoris is clefted, the labia are widely separated, and the urethra and vagina more or less defective. Eversion of the bladder is often complicated also by other malformations of the body, and in majority of instances leads to early death. Partial ectopia vesicæ offers a more favorable prognosis, particularly if a plastic operation is resorted to early. Temporary relief may be obtained from a suitable urinal held in place by means of a truss.

Prognosis bad.

MALFORMATIONS OF THE URETHRA, PREPUCE, TESTICLES, AND VAGINA.

Atresia Urethræ.—*Total atresia urethræ* is a rare malformation. When it does occur, it is usually epithelial in nature or at most membranous. In the former instance the atresia promptly yields to pressure with the tip of a sound, in the latter to a small incision and dilatation by means of a small, blunt silver probe.

Divulsion.

Complete absence of the urethra is extraordinarily rare.

Congenital stenoses are not rarely found along the urethra, and if presenting no distinct hindrance to urination are frequently overlooked.

In cases of marked urethral stenosis, the still patent urachus often permits the escape of urine through its fistulous tract running from the bladder to the umbilicus.

Misplacement of the Urethral Opening (Epispadias, Hypospadias).—The urethral opening may be situated on the upper part of the penis (epispadias) or at its inferior aspect (hypospadias). The latter abnormality is more frequent than the former. Both conditions are productive of more or less disturbance of urination (incontinence in epispadias; dysuria in hypospadias), secondary intertrigo, erosion and ulceration of the genitalia from the effects of the irritating urine, and later in life interference with virility.

Incontinence; dysuria.

Pronounced hypospadias (perineoscrotal) closely resembles hermaphroditism, and when it is associated with retention of the testicles it may be impossible to determine the sex of the infant.

Except in the very mildest cases early operative interference is indispensable.

Operative treatment.

CONGENITAL PHIMOSIS.

A moderate degree of adherence of the prepuce to the glans penis is physiologic in the newborn. Ordinarily the adhesions disappear spontaneously in the course of time. In some cases, however, the prepuce remains adherent and stenosed at its orifice so that the glans cannot pass through. In consequence there is more or less retention of urine between glans and prepuce (particularly if the latter is elongated or hypertrophied), infection and decomposition of the sebaceous secretion (smegma), and secondary inflammation of the penis and adjacent structures.

Dysuria. Retention of urine.

In the presence of inflammation urination is difficult and very painful; the infant cries, presses and strains (in predisposed children often the cause of hydrocele, hernias and prolapsus recti), or fearing pain retains the urine for many hours, a habit which is apt to give rise to cystitis, pyelitis, and even uremic convulsions.

Phimosis frequently forms also the cause of enuresis, priapism, masturbation, and a number of more or less reflex nervous phenomena.

[illegible]

In mild cases of phimosis the prepuce should frequently be pushed back and forth and the retained smegma removed. Where the adhesions are very firm they may be broken up with the aid of a dull probe and kept loose by daily pulling back the foreskin and applying an antiseptic cooling lotion such as lead-water or a 2 per cent. solution of aluminium acetico-tartrate. In this manner good results are obtained within a few days.

Lateral incisions and loosening of prepuce.

Where the preputial stenosis is the predominating trouble, slight nicking of the preputial ring with a scissors (laterally, above, and below), followed as before by loosening of the adhesions, daily preputial retraction and local antiphlogosis, is all that will be necessary to effect a permanent cure. This procedure is at all times preferable to circumcision, except in cases of phimosis associated with elongated or greatly hypertrophied foreskin and severe inflammation.

Circumcision, when indicated, should be performed under very careful aseptic precautions, preferably under general anesthesia. The surgeon grasps the prepuce between the thumb and index finger, exerting sufficient traction to draw it from the glans penis, puts over it a shield or forceps just in front of the glans, and with scissors or knife removes the distal, superfluous portion of the prepuce. He next seizes the inner layer of the prepuce, which still covers the glans, with a thumb forceps and with the aid of a scissors cuts it so far backward as to enable him fully to expose the glans and bring the edges of both preputial layers in apposition by a fine continuous suture. The dressing should consist of sterile gauze (not medicated! danger of intoxication). Numerous accidents have been reported as the result of circumcision, but all, except uncontrollable hemorrhage in the hemophilic, are preventable. In such hemorrhage the actual cautery should be resorted to without delay. Milder hemorrhages will often yield to firm compression of the penis with a hard catheter in the urethral canal.

Danger of intoxication, and hemorrhage.

CRYPTORCHIDISM.

(Undescended Testicles).

Normally the testicles descend into the scrotum by the end of fetal life. In the event of a constriction of the inguinal ring, and malformation of the testis, epididymis, or the vas deferens, etc., one (*monorchidism*) or both (*cryptorchidism*) testicles are not infrequently retained in the abdominal cavity, at the inguinal ring, or at the upper portion of the scrotum. More rarely the testicles become displaced, and through a false passage emerge either at the crural arch (crural testicle); under the fold of skin between the thigh and scrotum (scrotofemoral testicle); or behind the scrotum (perineal testicle).

In the majority of instances an undescended testicle is free from any serious consequences, and reaches its normal position spontaneously within the first few years of life. Occasionally, however, it may become impacted at the inguinal canal, giving rise to excruciating pain and inflammatory symptoms; if associated with a hernia, strangulation may take place in both structures at the same time; it may cause atrophy of the genitalia; it may be the seat of malignant degeneration, and, finally, it may be productive of a number of reflex phenomena (epilepsy?).

Spontaneous descent.

Strangulation.

Reflex symptoms.

Cryptorchidism should not be confounded with *anorchidism* or absence from the body of both testicles (is usually associated with rudimentary penis and, later, absence of spermatic secretion), or with ascent of the testicles from contraction of the scrotum (they descend with relaxation of scrotum).

Expectant plan of treatment up to puberty in the absence of complications. Capsular truss in cases of misplacement. Gentle

undescended
testicle

massage is useful. Orchidopexy and other surgical procedures as indications arise. Speedy operation in case of strangulation.

HYDROCELE.

It is a common affection of early infancy and most frequently congenital in nature. Varying with the seat of the accumulation

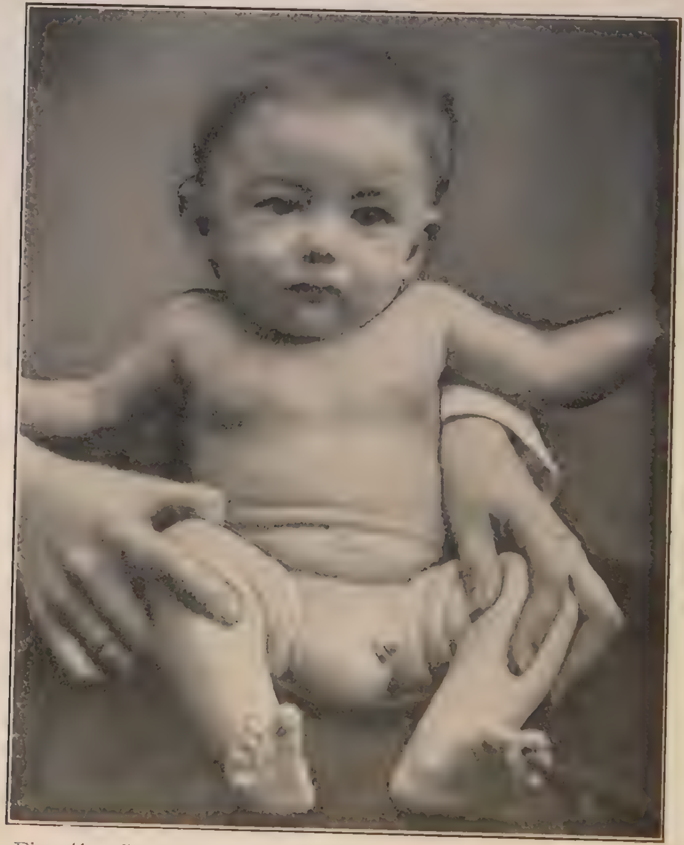

Fig. 41.—Congenital Hydrocele, Communicans. (*Sheffield.*)

of the abnormal quantity of serous fluid, we distinguish the following kinds:—

Swelling at lower part of scrotum.

1. *Hydrocele Tunicæ Vaginalis.* It is a unilateral, oval, smooth, translucent, more or less tense, fluctuating swelling, which appears first at the lower part of the scrotum, and gradually rises up to the abdominal ring. Posteriorly to the hydrocele usually lies the testicle.

2. *Hydrocele Funiculi Spermatici* (*hydrocele of the cord*), resembles the former, except that the testicle usually lies at the bottom of the scrotum and is distinctly separated from the hydrocele by a constriction. It is sometimes made up of several small cysts simulating a string of beads.

Separated from testicle by a constriction.

3. *Hydrocele Vaginalis Communicans* (*"Congenital Hydrocele"*). This form occurs when the tunica vaginalis preserves its communication with the abdominal cavity and becomes filled with serum, forming a cylindrical tumor, extending to and through the abdominal ring. It is often associated with hernia (hydrocele hernialis). As the contents of both are reducible on pressure the differential diagnosis between congenital hernia and hydrocele vaginalis communicans is sometimes difficult. In hydrocele, however, the return of fluid to the peritoneal cavity occurs without intestinal gurgling—the reverse being the case in congenital hernia.

Associated with hernia.

Hydrocele often disappears spontaneously, especially after removal of reflex irritation, *e.g.*, phimosis. If it persists, we employ local counterirritation (painting with tincture of iodin or mercury ointment), or aspiration, if the hydrocele enlarges. The latter procedure may be followed by the injection of a few drops of equal parts of tincture of iodin or carbolic acid and alcohol. Absorption of the fluid is hastened by a few large doses of potassium iodid. In hydrocele communicans a truss should be worn to prevent hernia. The pressure exerted will often obliterate the inguinal portion of the vaginal process, and also cure the hernia, if present.

Counter-irritation.

Truss.

If the aforementioned palliative and curative measures fail—which is rarely the case—a radical operation becomes necessary.

Radical operation.

Atresia Vulvæ.—It consists chiefly of a cellular adhesion of the labia minora, and may be partial or complete. In total atresia vulvæ there is anuria, with its secondary symptoms, necessitating immediate attention, *i.e.*, forcible separation of the labia with the fingers or with the aid of a dull sound or scalpel. In partial atresia separation of the labia occurs spontaneously.

Atresia Vaginæ Hymenalis (Imperforate Hymen).—This congenital malformation usually escapes observation until puberty, when partial or total retention of the menstrual flow gives rise to local and general disturbances.

Incision and packing with iodoform gauze readily remedies the trouble.

Atresia Vaginæ.—Like the aforementioned malformation, narrowing or complete closure of the vagina is not detected until after puberty. Total atresia vaginæ is usually associated with *absence of the uterus.* This should always be borne in mind before resorting to operative procedures for the relief of the atresia.

CONGENITAL MALFORMATIONS OF THE VERTEBRAL COLUMN

(Including those of the Sacrum and Coccyx).

SPINA BIFIDA (HERNIA OF THE CORD).

Meningocele Spinalis, Myelocystocele, Myelomeningocele.—Analogous to hernia of the brain (see "Cephalocele"), that of the cord also is divisible in three principal groups: Meningocele spinalis, myelocystocele, and myelomeningocele.

Filled with cerebro-spinal fluid.

(*a*) Meningocele spinalis is a protrusion of the pia mater without participation of the spinal cord. It is filled with cerebrospinal fluid, translucent, often pedunculated and may reach the size of a child's head. It is covered by normal skin. Paralysis is rare. Pressure on the tumor produces bulging of the fontanelles and spasms.

Solid masses and fluid.

(*b*) Myelocystocele is situated on a broad base and is readily replaceable on pressure. The covering skin is greatly distended but normal in color. Palpation reveals that the tumor consists of solid masses in addition to fluid. It is frequently associated with hydrocephalus and accompanied by motor and sensory disturbances.

Cord substance and its membranes.

(*c*) Myelomeningocele is a pear-shaped or spherical, fluctuating, tense, broad or pedunculated tumor the size of a walnut to that of a child's head. Its covering skin is bluish, very thin and traversed by numerous blood-vessels. It is composed of cord substance and its membranes and forms a true hernial protrusion through a cleft in the vertebral column. The cleft and to some extent also the hernial orifice can often be felt at the base of the tumor. Myelomeningocele is the most frequent variety of spina bifida and gives rise to marked motor and sensory paralyses.

Almost all forms of spina bifida are associated with hypertrichosis of the surrounding skin. This is especially pronounced, and indeed, often forming the only outward sign of deformity, in *spina bifida occulta* (a meningocele usually of the sacrolumbar region hidden under masses of fat). The hair is usually so arranged as to form a crown over the center of the defect. When well developed it may resemble a tail.

Pressure symptoms.

Apart from the malformation the condition of most children at first is perfectly normal. As the tumor enlarges the results of the pressure on the cord or the cauda equina gradually appear. The symptoms vary with the degree of involvement of the spinal cord; they are, therefore, most pronounced in myelomeningocele sacrolumbalis. Here we have motor and sensory paralyses of the legs, of the rectum, bladder, and the perineal muscles, convulsions and trophic disturbances. In less severe cases, the paralysis may be limited to the legs only.

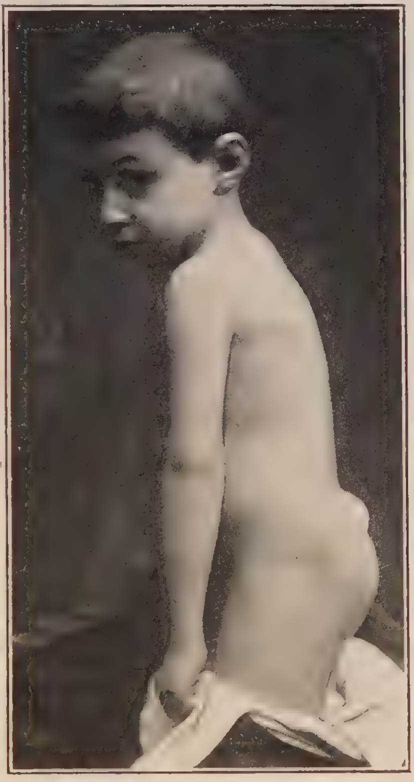

Fig. 42.—Spina Bifida in a boy 8 years old.

Differentiation from neoplasms.

Bearing in mind the characteristic symptomatology of spina bifida, *i.e.*, a more or less translucent, compressible, barely movable, thinly covered tumor, in the majority of instances associated with paralyses, there ought to be no difficulty in differentiating it from sacrolumbar neoplasms. In cases of doubt the diagnosis may often be cleared up by exploratory puncture and radiographic examination (the latter showing a vertebral cleft).

Spina bifida may sometimes escape notice when it is surrounded by a solid tumor.

The majority of children with marked spina bifida die when very young, often during birth, owing to rupture of the tumor and shock following rapid escape of the cerebrospinal fluid. Most of those who survive succumb later from rupture of the sac and subsequent infection and purulent meningitis; from gangrene and ulceration of the skin with subsequent sepsis; and finally, from intercurrent diseases and marasmus. Simple meningocele gives the best prognosis if recognized early and protected from external insults by a suitable pad or apparatus.

Protection against injury.

This palliative method of treatment should always be tried in cases of spina bifida that project very slightly and are covered by normal, well-nourished skin. Aspiration of the hernial sac is useful to relieve the symptoms of compression and to lessen the danger of spontaneous rupture. Aspiration may be followed by injection of iodin or preferably iodin-gelatin. In selected cases it may prove of permanent benefit.

Radical operation.

Radical operation is the ideal procedure in suitable cases. However, extensive paralyses, severe irreparable malformations elsewhere, hydrocephalus, and grave systemic affections are contraindications to operation. In such cases palliative and symptomatic methods of treatment are indicated.

CONGENITAL SACRAL TUMORS.

Closely related to and frequently associated with spina bifida (*q.v.*) are congenital sacral tumors. They may be classified as follows:—

1. **Double Formations**—
   - (*a*) Complete—two fully formed individuals grown together at the buttocks.
   - (*b*) Incomplete or parasitic formations—one or several rudimentary portions of the body attached to the buttocks of a fully formed individual.
2. **Sacral Hygromas.**—Single or multiple cysts, attached by a broad base to the dorsal surface of the sacrum. They are sometimes associated with spinal hernia.
3. **Tumores Coccygei.**—Neoplasms attached to the anterior surface of the sacrum and coccyx. The tumors are composed of fibrous or granular masses, generally of sarcomatous nature; sometimes of fat, cartilage, or bone. Occasionally they involve the spinal canal, or surround a spinal, dural protrusion (spina bifida). They never extend above the lower border of the gluteus, but spread toward the pelvis and between the legs of the child.

Spread downward.

4. **Caudal Formations**—
    - (*a*) Complete tails, manifested by an actual increase in the number of coccygeal vertebra.
    - (*b*) Imperfect tails, enlargement of vertebral column by rudimentary tissue.

But few children born with coccygeal tumors live beyond the age of one year. As the tumors enlarge, the infants succumb to progressive cachexia and exhaustion.

Protection against injury and infection.

As a rule, sacral tumors do not interfere with the life of the child if suitable protection is employed against vulnerability of the tumor and secondary infection. In some selected (see Spina Bifida) cases perfect results are obtained by skillful surgical interference.

MALFORMATIONS OF THE EXTREMITIES AND HIP.

Of the numerous malformations of the extremities (*e.g.*, complete absence; spontaneous partial amputations; fractures; supernumerary fingers and toes, etc.) but few are of interest to general practitioners, namely, congenital dislocation of the hip and club-foot. As these abnormalities are apt to be confounded with similar acquired affections, they will receive special consideration.

LUXATIO COXÆ CONGENITA

(Congenital Dislocation of the Hip).

The dislocation may be unilateral or bilateral. The acetabulum is rudimentary in form, and the head of the femur rests either above it, above and to the outer side, or above and behind it upon the ilium, sometimes immediately at the side of the great sciatic notch. If one leg is displaced it is shorter than the other, giving rise to distinct limping. If both sides are affected the gait is wobbling—"duck gait." As a result of this anomaly the buttocks project prominently backward while the spine is either thrown forward (lordosis, in bilateral) or tilted sideways (scoliosis, in unilateral dislocation). The differential diagnosis between this condition and rachitis and coxa vara is best established with the aid of the X-rays which shows the abnormal position of the head of the femur. If the malformation is detected early, it may be corrected either by opening the joint, replacement and fixation of the head of the femur in the artificially deepened acetabulum or by bloodless forcible reduction of the deformity

Wobbling gait; projecting buttocks.

Lorenz's operation.

and fixation of the head of the femur in the acetabulum by prolonged use of plaster-of-Paris bandages. For details of treatment the reader is referred to text-books on "Orthopedic Surgery."

TALIPES

(Club-foot).

1. Talipes varus, inversion of the foot, so that its sole faces the other foot. This is the most common of the congenital forms.

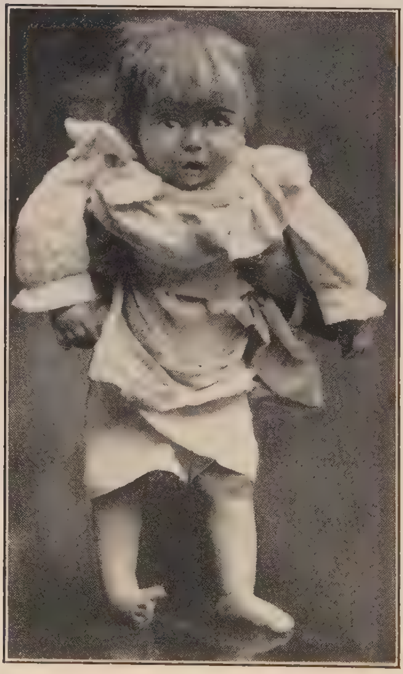

Fig. 43.—Congenital Talipes Varus. (*Sheffield.*)

2. Talipes valgus, flat-foot, effacement of the arch.

3. Talipes equinus, lowering of anterior part of the foot, the child steps on his toes.

4. Talipes calcaneus, elevation of anterior part of the foot, heel alone touching the ground.

Differentiation from rachitic and paralytic club-foot.

Compound forms may be produced by combination of the different varieties. The diagnosis of the type of club-foot can readily be made by inspection; it is sometimes difficult, however, to differentiate the congenital from the acquired forms, *e.g.*, rachitic or paralytic club-foot. (In rickets the distortion of the feet is generally associated with other pathognomonic symptoms of rickets and is gradual in development. In paralytic club-foot (*e.g.*, poliomyelitis) the limb is wasted, flabby and cold and there is a history of post-natal, gradual appearance often in association with other paralytic deformities.)

Congenital club-foot is being attributed to various causes, but is probably due to some mechanical interference with the normal development of the joints, ligaments or tendon insertions.

CHAPTER IV.

Birth Injuries.

Nature in its infinite wisdom provides a more or less large quantity of liquor amnii to protect the fetus in utero against undue pressure and possible injury. If, perchance, the amniotic fluid escapes prematurely either spontaneously or artificially, the fetus in its descent through the parturient canal, subjected to powerful pressure by the maternal structures or mechanical manipulations, sustains a number of injuries which vary in severity from simple external bruising to grave compound fractures and internal, sometimes fatal, injuries.

The effect of pressure.

I. SUPERFICIAL STRUCTURES.

CAPUT SUCCEDANEUM.

Vertex presentation being the most common form of delivery, the head consequently stands the brunt of the injuries. The so-called caput succedaneum is a circumscribed edema of the scalp. It is observed immediately after birth as a doughy, evenly distributed, variously sized tumor which disappears spontaneously by absorption, unless infected through external abrasions. In the latter event it requires surgical treatment, such as antiseptic drainage, incision and drainage.

Circumscribed edema.

CEPHALHEMATOMA.

More serious than the aforementioned condition is hemorrhage occurring between the pericranium and cranial bones in the form of a circumscribed, elastic, distinctly fluctuating, painless tumor, situated upon the right or left side of the head (sometimes both sides are affected). The cephalhematoma develops gradually within the first few days of extra-uterine life, and owing to the firm attachment of the periosteum to the edges of the cranial bones along the sutures, it never extends beyond the latter, or over the fontanelles. All around the tumor a hard, bony ridge is soon (after about two weeks) detected, which

Fluctuating tumor.

Bony ridge.

with the depressed center gives a sensation somewhat like that of a depressed fracture.

Differentiation from caput succedaneum, subcutaneous hemorrhage and congenital encephalocele.

Cephalhematoma may be mistaken for caput succedaneum, which appears immediately post partum and disappears after a day or two; for subaponeurotic or subcutaneous hemorrhages, which occur sometimes also from intrapartum pressure, but extend beyond the sutures; for congenital encephalocele, which lies between but not over the bones, pulsates, enlarges on crying or coughing, and can be partially reduced, and, finally, for vascular tumors, which are compressible and free from a bony ridge.

The tumor usually disappears spontaneously, sometimes requiring weeks and months to do it. If suppuration occurs, it calls for surgical interference.

HEMATOMA STERNOCLEIDOMASTOIDEI.

Hemorrhage within the sheath of the muscle.

Pathologically akin to cephalhematoma is the intrapartum hemorrhage which takes place within the sheath of the sternocleidomastoid muscle, as a result of rupture of several muscle fibers and consecutive myositis.

The tumor in the neck is generally observed a few weeks after birth, more rarely earlier. It varies in size from that of a hickory nut to a walnut. It is at first soft, later hard, cartilaginous in consistency. Severe hemorrhages may give rise to torticollis.

This condition demands perfect rest to the head, cold compresses for the relief of pain, and later gentle massage to promote absorption of the tumor.

℞ Ung. kalii iod. (U. S. P.),
  Adipis lanæ.....................................āā ʒij. | 8.
M. ft. ung.
Sig.: To be applied with gentle massage once a day.

II. DEEP STRUCTURES.

Birth traumatism is not always limited to the skin and muscles. Now and then the viscera (the lungs, liver, peritoneum, etc.), the bones, the peripheral nerves, the meninges and brain are involved. Fractures and dislocations are not rarely observed, especially in the long tubular bones and the clavicle, while the cranial bones are often badly displaced (the occipital and frontal are pushed under the parietals), fissured (see

Meningocele), compressed and fractured, giving rise to grave, frequently fatal, intracranial hemorrhages.

CENTRAL BIRTH PARALYSIS.

Cerebral Hemorrhage. Apoplexia Neonatorum.

Seat of hemorrhage.

Usually the seat of the hemorrhage is the subarachnoid space; often the delicate pia mater; sometimes between the dura and arachnoid; more rarely between the meninges of the cerebellum; the lateral ventricles, and exceptionally the brain substance.

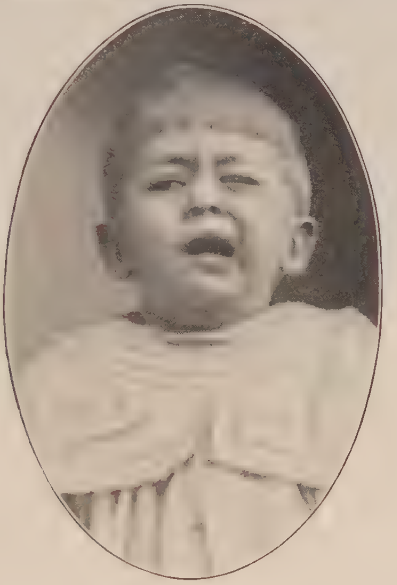

Fig. 44.—Obstetric Facial Palsy (15 months old). Failed to yield to treatment. (*Sheffield.*)

Symptoms of cerebral paralysis.

The symptoms differ with the extent and seat of the hemorrhage. Most infants are born asphyxiated. The majority of those born alive succumb within a few days under symptoms of asphyxia and atelectasis, slow irregular pulse, bulging of the fontanelles, convulsions, rigidity and paralysis. Those few who survive, at an early age present the symptom-complex of cerebral paralysis (see page 587), with or without idiocy.

The diagnosis of this condition in the absence of focal symptoms may present considerable difficulty. Nowadays it is greatly facilitated by lumbar puncture, the cerebrospinal fluid containing disintegrated blood-cells and products of decomposition.

Early operation.

The treatment is the same as for traumatic cerebral hemorrhage in the adult—principally surgical. Recent results warrant early surgical intervention.

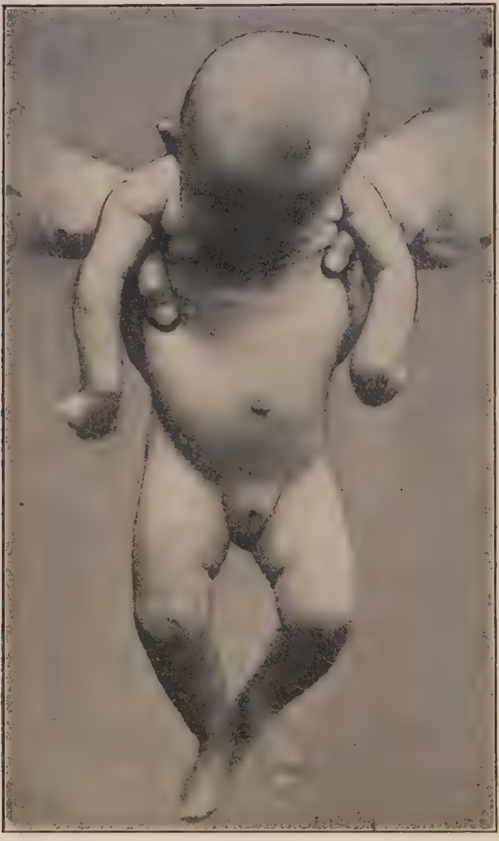

Fig. 45.—Bilateral Obstetric Brachial Paralysis, so-called "Duchenne-Erb's Paralysis." (*Sheffield.*)

PERIPHERAL BIRTH PARALYSES.

Facial Paralysis.

Rarely permanent.

Occasionally syphilitic.

Facial paralysis in the newly born is usually of traumatic origin, as a result of pressure exerted upon the facial nerve by the obstetrical forceps or deformed pelvis. It may be unilateral or bilateral. It resembles facial paralysis of older children (see page 541) except that it runs a milder course. Very rarely the paralysis is permanent. The so-called congenital, non-traumatic facial paralysis is probably syphilitic in nature.

Brachial Paralysis. Obstetrical Paralysis. Duchenne-Erb Paralysis.

In mild form it is of quite frequent occurrence.) In typical cases the paralysis is usually limited (80 per cent.) to the muscles supplied by the brachial plexus composed of the lower four cervical nerves and the first dorsal, and their branches *i.e.*, the deltoid, biceps, brachialis anticus, infraspinatus, supinator longus and the supinator brevis.

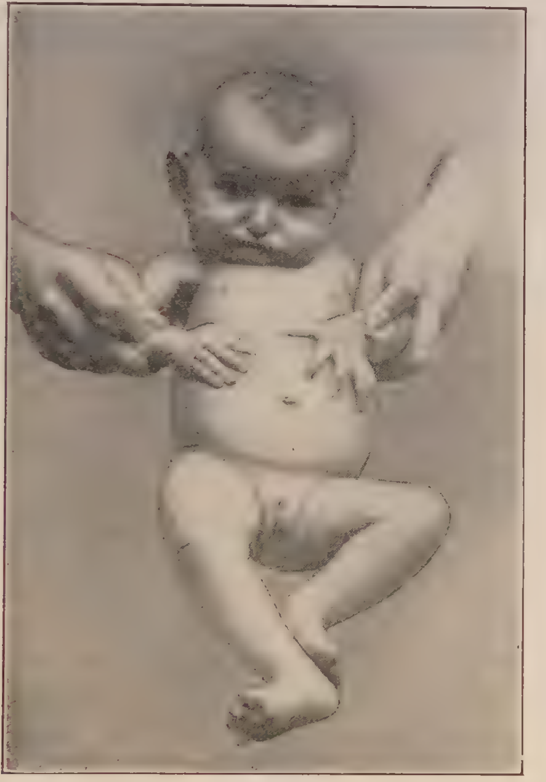

Fig. 46.—Bilateral Obstetric Brachial Paralysis (same as Fig. 45), six weeks later. Considerably improved. (*Sheffield.*)

(The arm (rarely both sides [see Fig. 45] are affected—from reckless instrumental manipulations) hangs motionless, the upper arm is rotated inward, the forearm is pronated, and the palm of the hand is turned backward and outward. The wrist- and finger-joints are usually only slightly affected; sensibility is intact and electrical reaction diminished or lost.

Usually unilateral.

Recovery is the rule in mild cases. Those lasting over three months show trophic changes in the affected muscles, especially the deltoid. The prognosis in cases of brachial paralysis presenting reaction of degeneration is doubtful.

Trophic changes.

After keeping the affected arm perfectly at rest for two weeks the faradic or galvanic current should then be applied daily, for about five minutes at a time, until muscular power has been restored. Gentle massage and passive motion are very useful as a prophylactic against atrophy and contractures. In complete rupture of one or more cords of the brachial plexus, nerve end to end anastomosis and tendon transplantation are the only curative means at our command.

Electricity and massage.

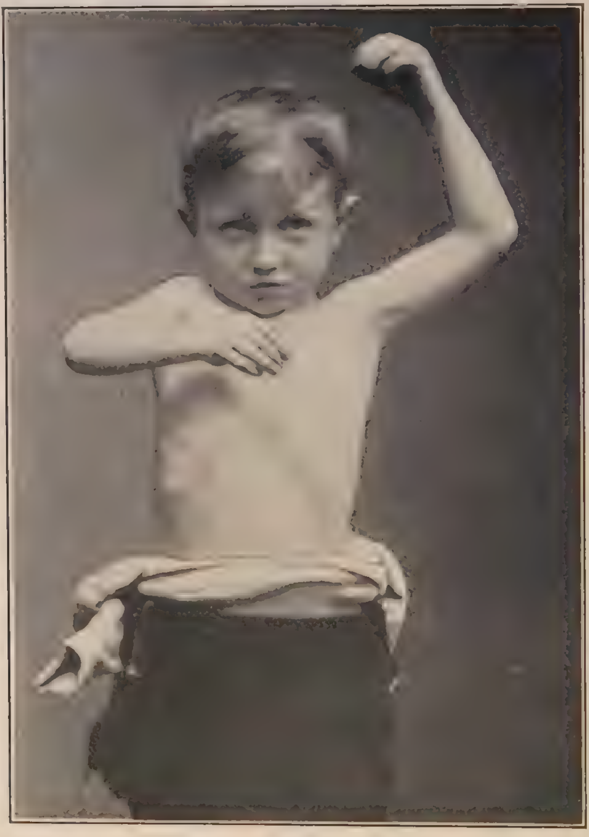

Fig. 47.—Obstetric Brachial Paralysis. Erb's "upper arm type." Failed to respond to treatment. (*Sheffield.*)

CHAPTER V.

Diseases of the Newly Born.

FEEBLE VITALITY OF THE NEWLY BORN.

THE physician is often confronted by a group of clinical phenomena in the newly born which may briefly be designated "feeble vitality." It is a clinical entity which, though greatly at variance as to cause and ultimate course, at birth presents a uniform symptom-complex and demands a more or less uniform mode of treatment.

It is characterized by pronounced respiratory and circulatory disturbances, subnormal temperature, somnolence, general debility and emaciation, and is usually associated with one or several presently to be described diseased conditions.

1. ASPHYXIA NEONATORUM
(Suspended Animation).

Asphyxia livida.

The asphyxia may be momentary, or last several minutes up to an hour or longer. Mild forms of asphyxia are manifested by slight lividity (asphyxia livida) of the face, feeble superficial breathing, and slow and weak heart-beat. If the asphyxia is allowed to continue, the face becomes deeply cyanosed and congested, the eyes bulge, the muscular tonus and cutaneous sensibility are retarded, the umbilical cord is collapsed, and respiration is barely perceptible. Finally, the infant becomes deathly pale (asphyxia pallida), the muscular tonus and reflexes are lost, the heart-beat is scarcely audible and respiration ceases.

Asphyxia pallida.

Post-mortem examination reveals overdistention of the right ventricle; cerebral, pulmonary and hepatic congestion; increased fluidity of the blood; serosanguinolent exudation in the serous cavities; accumulation of liquor amnii, blood and mucus in the air passages, and pulmonary atelectasis.

Artificial respiration.

Prompt and prolonged resuscitating efforts (Sylvester's, Schultze's and Laborde's) are usually attended by favorable results. However, intracranial hemorrhage with consecutive mental and physical defects are not infrequent sequelæ of severe forms of asphyxia.

2. ATELECTASIS NEONATORUM

(Congenital Collapse of the Lungs).

Normal inflation of the lungs.

Inflation of the lungs of the normal newly born infant begins with its first cry uttered announcing its arrival into the domain of the living. Succeeding respiratory acts gradually unfold the originally collapsed alveoli and bronchioles, and full expansion of the lungs is ordinarily completed within the first forty-eight hours. The posterior portions of the lower lobes, particularly the right, are last to expand.

Failure of the lungs fully to unfold gives rise to the condition under discussion, *i.e.,* atelectasis pulmonum.

Pathologic findings.

The alveoli and bronchioles are collapsed. The lung is brownish red in color, feels tough and resistant to the touch—like liver—does not crepitate, and sinks in water. Usually both lungs, particularly the posterior parts of the lower lobes, are affected. In cases succumbing to the disease after weeks or months there is also congestion of the heart, spleen and liver.

Occasionally caused by compression of trachea.

The causes of atelectasis are essentially the same as those of asphyxia; the former is sometimes a sequel of the latter, especially if inadequately treated. (Inflation of the lungs is occasionally interfered with by congenital hyperplasia of the thyroid or thymus glands compressing the trachea.)

Feeble respiration; cyanosis.

In marked atelectasis the infant makes but faint efforts to respire. It is pale, sometimes cyanotic; its temperature is subnormal, and its pulse slow and weak. It is unable to suckle properly and to cry aloud. It sleeps most of the time and but lazily responds to external influences. Auscultation discloses weak and vesicular breathing (never bronchial) and occasional crepitation. Slight dullness on percussion.

The great majority of otherwise healthy children recover under prompt and energetic treatment. Delicate infants either die a few hours, days or several weeks after birth from prostration following repeated attacks of cyanosis, or survive and remain debile for life, often suffering from organic defects, such as incomplete closure of the foramen ovale or ductus arteriosus, and the like,

Artificial respiration; stimulation.

The treatment of atelectasis consists in stimulating the respiratory and circulatory functions by keeping the infant wide awake; frequent change of position; artificial respiration; alter-

Valvular defects - Stenosis - Pulmonary artery - Aorta -

nating warm and cold baths or showers followed by brisk friction; oxygen inhalation and gentle faradization. Lustily crying babies do well.

3. VITIA CORDIS.

(See page 428.)

4. SYPHILIS EMBRYONALIS S. FŒTALIS.

The few babies who survive the syphilitic onslaught during intra-uterine life and are born at full term present a ghastly sight. They are shriveled and shrunken, emaciated and disfigured, with barely a spark of life in them. They are often asphyxiated and usually die soon after birth. Post-mortem examination reveals pronounced pathologic changes in the lungs (fatty degeneration of the pulmonary alveoli—"pneumonia alba"); in the liver (interstitial hepatitis); in the spleen and pancreas (induration and gummatous deposit); in the kidneys and suprarenal glands (perivascular infiltration and anemic necrosis); in the thymus gland (cystic degeneration and abscess formation); and in the osseous system (epiphyseal osteochondritis after multiple fractures). The skin affection consists chiefly of "pemphigus syphiliticus," a bullous eruption on a dusky red, slightly elevated base, with a sanguinopurulent content. It is usually localized on the palms of the hands and soles of the feet. Owing to extreme tenderness of the body (syphilitic myositis?) the infant is very restless, and cries pitifully when handled. (See Syphilis Congenita.)

Shriveled and shrunken.

Post-mortem findings.

Pemphigus.

5. PREMATURE BIRTH.

Children born before full term—between the twenty-eighth and thirty-eighth weeks of intra-uterine life—are designated "premature."

Thanks to the earlier and better recognition of syphilis, the more thorough appreciation of the methods of its prevention and cure, as well as the tendency of the syphilitic virus spontaneously to lose its virulence through attenuation, premature births, being due chiefly to parental syphilis, are no longer as frequent in occurrence as in former years.

Often caused by syphilis.

The physical condition of premature infants rests largely upon the period of prematurity, inherent vigor of the newly born, and the presence or absence of serious organic defects. Ordinarily premature infants are considerably punier than full term infants. They weigh and measure approximately:—

Before Birth—

AGE AT BIRTH.	WEIGHT.		SIZE.	
At 29 weeks	1600 Gm.	—3¼ ℔.	40 Cm.	—15 inches.
" 31 "	1900 "	4 "	43 "	16¼ "
" 33 "	2100 "	4¼ "	44 "	16½ "
" 35 "	2600 "	5¼ "	47 "	17¾ "
" 37 "	2800 "	5¾ "	48 "	18 "
" 40 " (full term)	3100 "	6¼ "	52 "	19½ "

Extremely low vitality.

The body is limp; the movements of the extremities are helpless and tardy. The face is usually sunken and senile. The skin is soft and delicate, vulnerable to an extreme, hence readily susceptible to infectious processes. Respiration is irregular, superficial and sometimes of Cheyne-Stokes type. Atelectasis and cyanosis are not rare accompaniments. The heart beat and pulse are weak, often irregular, and the blood lacks in coagulating power. The bones are soft, more or less yielding to light manipulation. The temperature is subnormal. Premature infants, as a rule, are unable to suckle or swallow properly, and owing to incapacity of the digestive organs and atony of the intestinal musculature, to fully assimilate the food consumed. Severe colic and uric acid infarcts, which latter often lead to anuria and other uremic manifestation, add misery to their painful existence.

High mortality.

Encumbered with so numerous deficiencies, the span of life of the delicate premature infant must obviously measure but a few hours or days. The mortality of premature infants under 1600 grammes in weight, especially if they are inadequately cared for, is estimated to be about 80 per cent.; of those weighing over 2000 grammes, 40 per cent.; while of those weighing over 2500 5-6 grammes only 20 per cent.—almost as low as with full-term babies. Such as survive, however, often remain very feeble for many years, manifest a greater tendency to disease, and lack power of resistance to overcome it. (Occasionally, after many ups and downs, premature infants marvelously extricate themselves from the pangs of death and grow up full of vivacity and vigor.)

Recovery under suitable treatment.

It is therefore incumbent upon the physician to look upon every premature infant that respires at birth as one whose life can be preserved by suitable care and treatment.

Management of "Feeble Vitality of the Newly Born" with Especial Reference to the Premature Baby.

Three special indications are to be met in the management of newly born, delicate infants. We must (1) endeavor to maintain the best features of antenatal life; (2) supply nutriment suitable for the infant's growth and development and (3) awaken and strengthen the dormant or inefficient functions of its organs.

Artificial heat.

The first prerequisite should be met by an artificial environment which should as nearly as possible resemble that of the interior of the uterus.

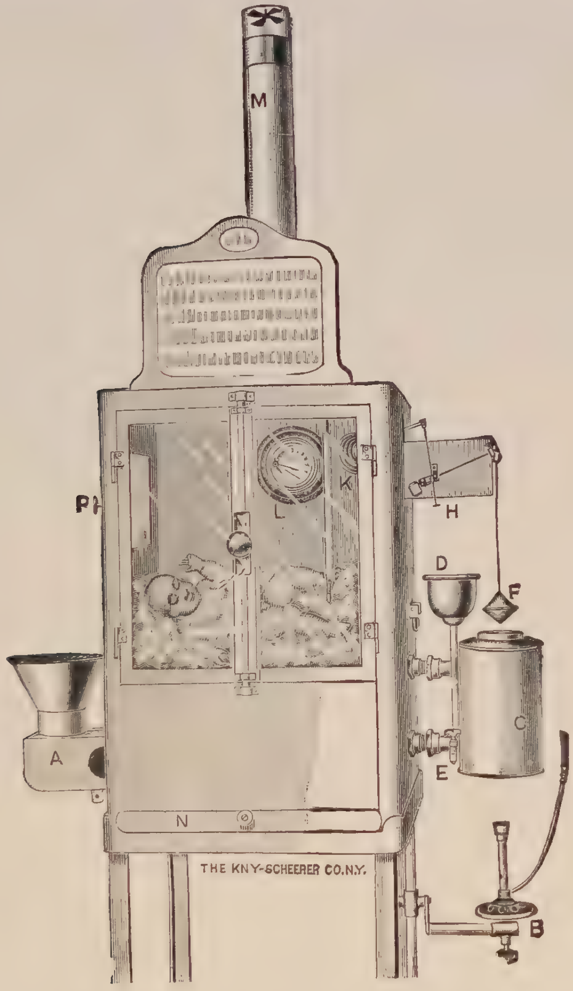

Fig. 48.—Incubator for Premature Infants.

Incubator.

The numerous modern incubators on the market in many instances answer the purpose. The infant is clothed in a woolen shirt and napkin and placed in the incubator, upon a cotton bed.

The temperature of the incubator is maintained steadily at about 96° F., and fresh air supplied by the automatic ventilating contrivance and by off and on leaving the door open. Infants showing a fair amount of vitality usually get along very well without incubators, the latter being supplanted by ordinary bassenets and warm-water bags. Delicate incubator babies should be disturbed as little as possible, and removed only for feeding and cleansing (by means of lukewarm oil), or for such therapeutic purposes (*e.g.*, artificial respiration) as necessity arises.

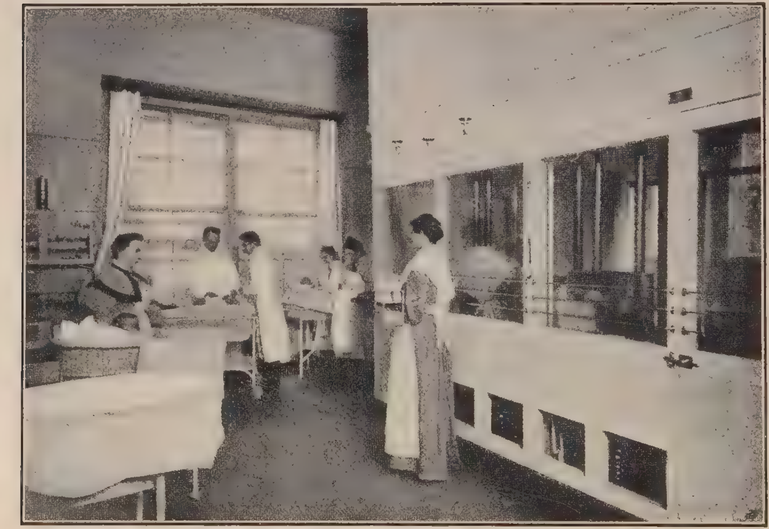

Fig. 49.—Incubator Room for Newly Born Babies with Feeble Vitality. Prof. Th. Escherich. (*Sheffield.*)

Avoidance of unnecessary handling.

Bathing is contraindicated, and any undue handling of the skin or mucous membranes must be carefully avoided, since most trifling injuries are very apt to be followed by fatal sepsis.

Every effort should be made to feed the premature infant on woman's milk. for at least the first few weeks of extra-uterine life. When too weak to suckle from the breast, the milk may be given in diluted form (1:2) by means of a dropper or little spoon, care being taken that the milk flows down into the throat very slowly, lest it enter the trachea and lead to aspiration pneumonia. In the absence of breast milk, light mixtures of cows' milk (½ per cent. of fat, ½ per cent. proteids and 5 per cent. of milk-sugar) should be administered every hour or two, in quantities of 1 to 4 teaspoonfuls.

Careful feeding.

Stimulation.

The third indication applies principally to infants who, though born at full term, possess very little vitality, and whose organs, especially the heart and lungs, fail to functionate. This vitality is best aroused by artificial respiration—by alternate flexion and extension of the infant's body while it lies on the operator's palms. An occasional dash of cold water upon its face, to induce the child to cry aloud and take deep breaths, and stimulation by means of oxygen and strychnine, serve as useful adjuvants.

SCLEREMA NEONATORUM

(Sclerema Adiposum).

This very rare affection may be primary, without any apparent cause, or secondary in nature, as a result of great loss of body fluids (internal hemorrhages, gastrointestinal disease), or extensive exudations into internal cavities (thorax). It occurs principally in the premature, very feeble and badly nourished infants in the first few days of life (but also very much later, up to six months of age).

Begins with lower extremities.

It begins in the lower extremities, particularly the calves. From here it spreads symmetrically over the thighs, loins, trunk, neck, upper extremities and head, leaving penis, scrotum, planta pedis and palma manus uninvolved. The skin is dirty yellow, very tense, cold, hard, immovable over the underlying structures, and does not pit on pressure.

Marbleized.

Interference with suckling.

Subnormal temperature.

From day to day the skin becomes more indurated, marbleized, and the patient lies stiff with rigid, mask-like face and firmly closed mouth as though in a state of tetanus. Suckling is often impossible. There is gradual sinking of all vital functions. The temperature falls (to 85° F. or lower), the heart action becomes weak, the pulse is slow and barely perceptible, respiration shallow and irregular, the voice feeble and whining, the intestines and kidneys are inactive, the child wastes rapidly, and death ensues in about a week from exhaustion or some complication, the commonest being pneumonia and sepsis. Milder cases, especially older infants, not infrequently recover.

Active stimulation.

Early hypodermo- and entero-clysis with hot (104° to 106° F.) normal saline solution (from 2 to 3 ounces t. i. d.); gentle massage with oil; stimulation; maintenance of body heat; careful feeding, etc., as outlined under "Feeble Vitality of the Newly Born." (See page 168.)

SCLEREDEMA NEONATORUM

(Sclerema Serosum).

Chest usually free.

This form of edema affects especially premature, weak (twins), atelectatic, and syphilitic infants. It usually begins a few days post partum (it is rarely congenital) with puffiness and swelling of the feet and legs. The edema soon extends upward (involving also the mons veneris, scrotum and penis) over the entire body, except the chest, and rarely the eyelids and face. The skin is tense, shiny, waxy white, or cyanotic, and pits on pressure. When the edema increases it greatly resembles true sclerema, but may be differentiated from the latter by bearing in mind the following characteristic symptoms:—

Differentiation from sclerema.

	Sclerema.	Scleredema.
Color of skin..........	Dirty yellow.	Shiny or mottled.
Parts exempt...........	Genitals, palms of the hands and soles of the feet.	Chest.
Pitting on pressure.....	Absent.	Marked.

The general symptoms, such as low temperature, great depression, etc., are not quite as pronounced as in sclerema adiposum.

The prognosis is not as grave as in true sclerema.

Stimulation; diuresis.

The treatment consists chiefly of stimulation (camphor, digitalis), hot baths, massage and passive motion, active diuresis and proper feeding. See also "Feeble Vitality of the Newly Born."

SEPSIS NEONATORUM.

With the usual aseptic precautions that are now being taken in the management of labor and the puerperium, the number of cases of sepsis neonatorum has been reduced to a minimum. This is true especially of systemic sepsis. The extreme importance, however, of the subject in question, demands its careful consideration.

LOCAL SEPSIS.

Omphalitis (Inflammation of the Navel).

Local inflammation.

Simple omphalitis is manifested by delayed closure of the umbilical wound after separation of the umbilical cord, wetness, slight suppuration, and incrustation. There is no inflammatory reaction in the surrounding parts. The general health is undisturbed.

Phlegmonous omphalitis usually begins the second week after birth. The navel forms an ulcerated conical projection. The surrounding tissue is firm, infiltrated, glossy and painful to the touch. Sometimes the inflammation extends rapidly over the abdominal wall or into the deeper structures, giving rise to peritonitis. The constitutional symptoms vary with the degree of severity of the affection, but are sufficiently pronounced as to make the child quite ill and to render the prognosis doubtful. Milder cases often terminate in suppuration and with careful treatment (see page 174) in recovery.

Ulcerated conical projection.

Constitutional symptoms.

Erysipelatoid omphalitis is a very grave affection, often terminating fatally either within a few days from exhaustion or a week to ten days later from septic peritonitis, icterus, and local suppuration. The symptoms and treatment are the same as in ordinary erysipelas.

Septic symptoms.

Diphtheritic omphalitis (ulcus umbilici) is characterized by a fibrinous umbilical exudation which when cast off leaves behind a superficial or deep ulcer. Occasionally it is due to Klebs-Löffler bacillus.

Klebs-Löffler bacilli.

Gangrenous omphalitis ends fatally in the majority of cases. At first a small, discolored, ulcerated spot, if not immediately arrested, it rapidly develops into a large, gangrenous, fetid mass. It sometimes extends into the deeper structures, giving rise to peritonitis, urinary and fecal fistulæ, profuse hemorrhage and pronounced constitutional symptoms.

Sloughing.

As the umbilical wound forms the principal and most frequent portal of entry for septic infection, the importance of caring for the umbilicus with the minutest detail is quite obvious. Strictest cleanliness should be enforced and unnecessary handling prohibited. Clean scissors, clean ligature, preferably composed of several strands of cotton or silk thread, and, above all, clean hands should be used in cutting, ligating, and dressing the cord. The dressing should consist of a few layers of sterile linen cloths and a dusting powder (1 part of salicylic acid and 6 parts of starch) and be changed every alternate day, preceded by cleansing the wound with a little pure alcohol to hasten desiccation of the umbilical rest. As moisture favors the growth and absorption of the bacteria which accumulate at the navel wound, the child should daily receive a sponge-bath instead of a tub-bath, until the navel has completely cicatrized.

Aseptic precautions.

Dry dressing of navel.

To prevent hernia as well as access of dirt, the umbilical band should be continued for some time after complete healing of the navel.

Early and energetic treatment.

If inflammation of the navel, no matter how slight in degree, occurs notwithstanding all the precautions, it should receive immediate and energetic treatment. Procrastination is dangerous, nay, often fatal.

Nitrate of silver.

Cauterization of the affected parts with a 2 per cent. to 5 per cent. solution of nitrate of silver, once a day or less often, is very useful in all forms of omphalitis. The wound should be kept scrupulously clean, and protected by a moist (boric acid solution 4 per cent.) gauze dressing, covered by rubber tissue. If the septic process does not yield to this treatment early, a surgeon should be consulted. A bacteriologic examination may prove helpful in giving a correct clue as to the treatment, as for example, in diphtheritic omphalitis, where diphtheria antitoxin is of undoubted benefit. (See also "Biologic Therapeutics," page 94.)

Diphtheria antitoxin.

Omphalorrhagia (Bleeding from the Navel. Idiopathic Umbilical Hemorrhage).

Umbilical hemorrhage may occur as a result of tearing of the cord during delivery, defective ligation, or imperfect establishment of respiration (delaying the closure of the umbilical vessels). The hemorrhage may be slight or severe, but is readily controllable. In contradistinction to these forms of navel bleeding which take place soon after birth, there is another variety of bleeding from the navel, the so-called "Idiopathic or Spontaneous Umbilical Hemorrhage" which occurs at about the time the umbilical rest separates (between the fourth and ninth days). The bleeding takes the form of a steady oozing of blood as though coming from a compressed wet sponge. It is probably due to sepsis of the umbilical blood-vessels. Some authors are inclined to attribute it to congenital syphilis or transitory hemophilia (see page 477). In a great many instances the hemorrhage cannot be arrested, death taking place either from exsanguination or from gradual exhaustion and complications (sepsis).

Sepsis of umbilical blood-vessels.

Syphilis. Hemophilia.

For details of treatment see "Hemorrhea Neonatorum" (page 181).

Umbilical Granuloma (Excrescence, Fungus, Sarcomphalos).

Strawberry-like tumor.

It is a strawberry-like, small tumor, attached to a broad base or pedicle at the umbilical stump. It bleeds readily and usually discharges thin pus. Like exuberant granulations in other localities, it is promptly cured by a few applications of nitrate of silver (the stick or 10 per cent. solution). It should not be confounded with "Persistent Omphalomesentericus." (See page 146.)

Nitrate of silver.

Ophthalmoblennorrhea Neonatorum (Gonorrheal or Purulent Ophthalmia).

Gonorrheal ophthalmia is caused by infection of the conjunctiva of one or both eyes by the gonococcus (Neisser). The inoculation usually occurs during the passage of the head through the parturient canal containing a gonorrheal discharge. It may also be conveyed to the eyes of the infant post partum by means of the fingers of the attendant or articles in use which have been soiled by the purulent discharge.

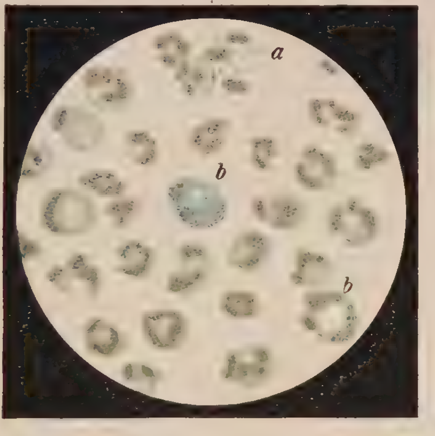

Fig. 50.—Gonococcus. (Gonorrheal Pus.) Stained one-half minute with methylene-blue. *a*, Free in groups. *b*, Enclosed in pus cells. Leitz ocular I. Oil immersion 1/12. (*Lenhartz* and *Brooks*.)

Gonorrheal infection.

The disease begins two or three days after the gonorrheal inoculation with intense tumefaction of the lids, redness, swelling and thickening of the conjunctivæ, lacrimation, and mucous and mucopurulent secretion. From day to day the discharge becomes thicker and more purulent; the conjunctiva assumes a velvet-like appearance (chemosis), and papillary deposits or longitudinal folds appear upon the conjunctiva bulbi. If not immediately arrested, especially if the purulent secretion is

Thick, purulent discharge.

allowed to accumulate between the edematous, pasted lids, the disease spreads rapidly to the cornea, causing haziness, maceration and partial or total perforation. As a result of the latter, and depending upon its location total or partial staphyloma, panophthalmitis with phthisis bulbi, capsular cataract, and anterior synechiæ may supervene.

Involvement of cornea.

Occasionally, particularly in delicate infants, gonorrheal conjunctivitis gives rise to numerous complications, such as articular affections, gonorrheal rhinitis, stomatitis, etc.

The duration of the disease varies from four to eight weeks.

Until the introduction of Credé's method of prophylaxis, gonorrheal ophthalmia was supposed to have contributed 60 per cent. of the cases of blindness of one or both eyes. At present the percentage has been reduced to one-third and with early and careful treatment the prognosis is still more favorable.

Gonorrheal ophthalmia is not to be confounded with the simple conjunctivitis not infrequently met in the newly born in connection with local sepsis. The latter variety is readily recognized by the absence of gonococci in the discharge and by its much milder course. Where there is the least suspicion of gonorrhea in the mother, her parturient canal and external genitalia should be carefully disinfected by a bichlorid solution (1 to 5000) before, during and after delivery. In addition to this the following directions given by Credé in the way of prophylaxis should promptly be resorted to: Wash off each eye with a boric acid wipe; into each eye instill two drops of a 2 per cent. solution of silver nitrate; in about thirty seconds wash out the excess with saline solution. This should be done as early after birth as possible. During the puerperal state the child should be kept away from the mother. In absence of gonorrhea, the infant's eyes should be washed with a saturated solution of boric acid.

Credé's method.

If only one eye be infected the fellow-eye should be securely covered by a watch-glass or a small pad of lint, oiled silk and roller bandage. This protected eye should be inspected and cleansed twice daily.

Protection of healthy eye.

As soon as the child is seen by the physician he should pencil the affected eye with a 2 per cent. silver solution. If this occurs early, the ophthalmia may sometimes be arrested in its incipiency or at least rendered milder in its course.

Nitrate of silver.

The affected eye must be handled by the nurse from behind the patient's head. Small, round layers of lint are transferred

Ice cloths.

every three to five minutes from a large square of ice to the affected eye, continuously for one hour. An intermission of one hour is then given and the cold applications are resumed. This should be continued day and night until there is positive evidence of abatement of the inflammation and excretion. This usually occurs within two weeks. The eyes should be carefully but very gently cleansed every half an hour with warm saturated solution of boric acid (4 per cent.). If the lids are so swollen as not to permit thorough cleansing, canthotomy may be resorted to. Silver being the most proficient antigonococcus, a 2 to 3 per cent. solution should be applied to the conjunctiva daily as long as the excretion is profuse and less often when it becomes more scanty and less purulent. In involvement of the cornea the ice-cloths should be discontinued, but not the silver applications. A 1 per cent. solution of atropine should be used as necessity arises.

Gentle cleansing.

Surg 11 or [illegible] [illegible] by [illegible] [illegible]

Examination of the discharge for gonococci should be made at least once a week, and the case should not be regarded non-contagious and out of danger until the discharge from the eye remains free from gonococci for at least two weeks. The treatment of gonorrheal ophthalmia should not be intrusted to unskillful hands. The better trained the nurse is in handling serious eye cases, the more rapid and perfect the recovery.

Repeated examination of discharge for gonococci.

Pemphigus Neonatorum.

Simple, non-syphilitic (see page 400) pemphigus makes its appearance between the fifth and twentieth day of the child's life. It is quite communicable, sometimes epidemic, and is probably due to the staphylococcus pyogenes aureus. Its seats of predilection are the abdomen and inguinal region, but the lesion may be found on any part of the body. It but very rarely affects the palms of the hands and soles of the feet—herein markedly differing from syphilitic pemphigus. The eruption consists of tense bullæ, varying in size from a lentil to a quarter of a dollar piece and contains a serous, rarely seropurulent fluid. The blebs are situated upon a reddened base, and on bursting leave moist red spots which very soon are covered over by skin. Occasionally ulceration of the skin supervenes, and is accompanied by high fever and other constitutional symptoms (malignant pemphigus). This severe form of the disease is

Communicable.

Palms of hands and soles of feet usually free.

Malignant pemphigus.

observed particularly in cachectic and bottle-fed infants, exposed to unsanitary surroundings, and often leads to fatal issue. In otherwise healthy, well-nourished and well-kept infants, recovery may be expected within from two to three weeks.

Isolation.

Simple pemphigus is preventable by strict attention to general hygiene and proper feeding. Those in charge of the child should be cautioned as to the communicability of the disease. If large surfaces are involved warm baths are very useful, preferably with oak bark (quercus corticis), bran or clay. They may be administered two or three times a day and followed by dusting over the moist surface

℞ Bismuthi subgall.,		
Acidi salicyl.	āā gr. x	0.6
Zinci stearat.	℥j	30.

and enveloping the body in cotton. Occasionally, application of a 2 per cent. solution of nitrate of silver.

Dermatitis Exfoliativa Neonatorum.

Rhagades.

Large lamellæ.

Slight dermatitis, or erythema, with or without desquamation, is more or less physiologic in the newly born. There is, however, an obscure (sepsis?) form of exfoliative dermatitis which is peculiar to early infancy (usually in the second, rarely after the fifth week of life), and is closely related to pemphigus. It begins with inflammation of the oral mucous membrane, rhagades at the angles of the mouth and diffuse redness of the entire body, followed by active desquamation of the skin in large lamellæ. It is sometimes preceded by detachment of skin and bursting of vesicles filled with clear fluid. Not infrequently the erosions extend to the oral mucous membrane.

Bad prognosis in delicate children.

The disease runs its (afebrile) course in a few weeks, and in robust children ends favorably. In delicate children it may be followed by general furunculosis or even gangrene, gastrointestinal disturbance and pneumonia and prove fatal.

Like non-syphilitic pemphigus, dermatitis exfoliativa is preventable by scrupulous cleanliness, and avoidance of local irritation. The local treatment consists of inunctions of 1 per cent. salicylic or carbolic acid oil.

GENERAL SEPSIS.

In speaking of primarily local septic affections attention has been directed to the frequency with which grave constitutional symptoms are observed during their protracted course. In these cases the systemic manifestations are secondary to the local ones, and if the latter are detected and treated early, the former may be prevented or arrested in their incipiency. We are now about to describe a group of septic diseases in the newly born which either present no visible local lesions at all, or so slight as to escape attention in their early stages.

Prophylaxis.

Tetanus (Trismus) Neonatorum.

Tetanus in the newly born, like the corresponding disease in the adult, is due to the tetanus bacillus (Nicolaier, Kitasato).

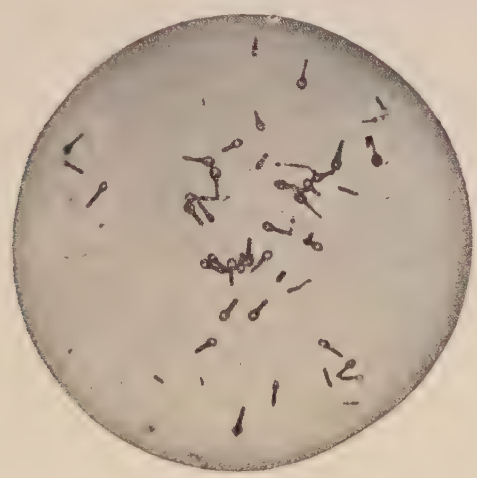

Fig. 51.—Bacillus Tetani. × 1000. (After *Fränkel* and *Pfeiffer*.)

Infection usually occurs through the umbilical stump or circumcision wound. The bacillus multiplies by spore-formation and generates toxins which enter the system, and are absorbed principally by the endings of the motor nerves. From here the toxins are ultimately carried to the anterior horns of the spinal cord and the nuclei of the medulla oblongata—hence the tetanic contractions.

Infection through open wound.

The symptoms begin within the first week after birth, rarely later (after ritual circumcision), with restlessness, dropping of the nipple of the breast or bottle with a cry, and tension of the masseters. The spasm rapidly involves the orbicularis oris and palpebrarum muscles, the lower jaw becomes rigid, the mouth

Lockjaw.

proboscidiform, the forehead and cheeks are wrinkled, and the eyelids are half closed (risus sardonicus). The hands are clenched, the legs flexed and abducted and, varying with the degree of severity of the attack, there is more or less marked opisthotonos. At first the paroxysms occur only during the act of nursing, gradually, however, more frequently and more persistently. In severe cases there are also spasmus of the glottis, of the esophagus, and diaphragm, and in consequence attacks of asphyxia which may end fatally. On the other hand, the affection may run a protracted course, sometimes for weeks, and end occasionally in recovery.

Spasmodic contraction of extremities. Opisthotonos.

The more violent the attacks and the higher the temperature, the less favorable the prognosis; 70 per cent. of the cases succumb within a few days, either from spasm of the diaphragm or, more rarely, from exhaustion.

Asepsis.

Careful protection against wound infection and prompt attention to existing traumatism. Considering the very grave prognosis under the ordinary methods of treatment and the occasional success obtained by means of hypodermic or subdural administration of tetanus antitoxin, the latter should be resorted to at the earliest possible time, either as a prophylactic immediately after the injury (1500 units) or as a curative measure (3000 units p. r. n.), in addition to the symptomatic treatment generally in vogue. This consists of perfect rest, lukewarm baths, chloral hydrate and the bromides per rectum, feeding (mother's or diluted cows' milk) with a tube through the nose, and avoidance of any irritation of the skin.

Tetanus antitoxin.

Arteritis and Phlebitis Umbilicalis.

This condition is usually observed secondarily to omphalitis, but may occur as a primary disease. In the latter event no local alterations are discernible at the navel and the grave affection frequently escapes notice until pronounced symptoms of general sepsis make their appearance. These consist of restlessness, fever, prostration and death within a few days, or gradual exhaustion from numerous complications. In umbilical phlebitis intense icterus—from extension of the inflammation to the liver—forms a characteristic symptom. In some cases of arteritis and phlebitis umbilicalis a fistulous tract is observed at the navel which on pressure discharges blood and pus containing pathogenic micro-organisms.

Usually secondary to omphalitis.

Intense icterus.

For prophylactic and local treatment see "Omphalitis" (page 172). The constitutional symptoms call for symptomatic treatment. Thus, careful feeding, preferably breast milk; active stimulation by means of enteroclysis, hypodermoclysis, sterile camphorated oil, etc. Antistreptococcic serum is deserving of trial.

Antistreptococcic serum.

Erysipelas Neonatorum.

This affection begins suddenly, with high fever, convulsions, and often other symptoms of general sepsis. The glossy redness rapidly extends over large areas, often over the entire body. The disease proves fatal in a few days and the cases that survive the acute attack usually succumb to cutaneous necrosis (particularly of the scrotum, extremities), copious diarrhea, septic peritonitis, pneumonia and exhaustion.

Rapidly spreading glossy redness.

The treatment is principally prophylactic. The inflamed areas should once a day be painted with pure ichthyol.

HEMORRHEA NEONATORUM ACQUISITA.

Melena Vera. Epidemic Hemoglobinuria with Icterus (Winckel's Disease). Acute Fatty Degeneration (Buhl's Disease).

Latest investigations tend to establish the fact that the aforementioned symptom-complexes in all probability are part-manifestations of general sepsis of the newly born. Mention, however, may be made that congenital heart disease, syphilis, and "feeble vitality" serve as predisposing causes.

Symptoms of general sepsis.

1. Melena Neonatorum.

Melena *vera* should not be mistaken for melena *spuria,* in which condition the blood originates from erosions in the mouth or nasopharynx, or from swallowing of blood from fissured nipples, etc.

True and false.

Melena vera usually begins in the first few days of the child's life with bleeding from the bowels, and often with hematemesis. As a rule, the blood is mixed with stool, and is dark brown or black in color. In some cases the loss of blood is slight, recurs at long intervals and terminates spontaneously without serious consequences except tedious convalescence. In the majority of cases of genuine melena, however, the bloody discharge is profuse and leads to rapidly increasing anemia and collapse.

Blood from alimentary canal.

2. Epidemic Hemoglobinuria with Icterus in the Newborn (Cyanosis Icterica cum Hemoglobinuria, Winckel's Disease).

This extremely grave (90 per cent. mortality) epidemic affection makes its appearance about the fourth day post partum, in apparently healthy-born and well-developed children. The infant becomes restless, refuses nourishment, shows signs of respiratory disturbance and slight rise of temperature. The skin turns greenish-yellow, and soon deeply jaundiced and cyanotic. Collapse, somnolence and convulsions, rarely preceded also by vomiting and diarrhea (no blood), are rapidly followed by death. The urine is pale brown, contains hemoglobin, renal epithelium, granular and blood casts, and masses of detritus, but no free blood-corpuscles.

Hemoglobin, but no blood-cells in urine.

The autopsy reveals congestion and fatty degeneration of the internal organs, with punctiform hemorrhages, especially in the mucous and serous membranes; masses of granular hemoglobin in the kidneys and spleen, thickening of the blood, and slightly increased leucocytosis.

Pathologic findings.

3. Acute Fatty Degeneration of the Newborn (Buhl's Disease).

The essential anatomical features of this rare but very malignant affection are fatty degeneration of the internal organs, notably the heart, liver and kidneys, and hemorrhages in any of the viscera, and into the serous cavities.

Fatty degeneration of internal organs.

The disease attacks full-term infants who for some inexplicable reason are born asphyxiated. Those few who survive, respire badly, are cyanotic, or rather icteric, and present hemorrhages in the skin and mucous membranes, from the alimentary canal, and the umbilicus. They almost invariably succumb before the end of the second week from progressive anemia, anasarca, and collapse.

Asphyxia. Hemorrhages.

Treatment.—The indications for the treatment of any of the manifestations grouped under "Hemorrhea Neonatorum Acquisita" are: 1, to arrest the hemorrhage; 2, to improve or, at least, maintain the vitality of the newly born infant. The first indication may be met by the administration of sterile (!) warm gelatin subcutaneously (10 per cent. solution, in doses of a quarter to half an ounce three times a day), per rectum (one to two ounces) and by mouth (2 per cent. to 5 per cent. solution, a teaspoonful t. i. d.), in addition to local hemostasis by means

Gelatin.

of adrenal solutions, perchlorid of iron, packing, compression, and cauterization (by nitric acid, Paquelin cautery). Very recently good results have been reported from hypodermatic injection (once or twice) of 5 cubic centimeters of fresh rabbit-serum.

Fresh rabbit-serum.

To meet the second indication, the reader is referred to the instructions given under the "Management of Feeble Vitality of the Newborn" (see page 168).

FUNCTIONAL DISORDERS OF THE NEWBORN.

URIC ACID INFARCT, ICTERUS, MASTITIS.

Uric Acid Infarct.

The urine of the newly born is clear immediately after birth, but turns turbid soon after and remains so for the first four or five days. It contains bladder and kidney epithelia, hyaline and epithelial casts, and a large quantity of urates. In consequence of the sudden alteration in the blood circulation there is an excessive excretion of nitrogenous metabolic products, and, as the newly born consumes but very little water during the first few days of life, uric acid crystals and ammonium urate instead of being washed away are retained in the renal tubules.

Uric acid crystals.

The symptoms accruing from this functional insufficiency depend greatly upon the degree of obstruction of the urinary tubules. Ordinarily gradual elimination of the uric acid and ammonium urate crystals occurs within a few days without any abnormal manifestations, except restlessness and crying just before and during the act of urination, and passage of small quantities of highly colored urine showing brick-red stains and a fine granular deposit on the diaper. Occasionally, however, there are complete retention of urine, fever, and, owing to irritation of the renal pelvis, nephritis with its concomitant symptoms (albuminuria neonatorum).

Anuria. Complications.

Treatment.—Large quantities of fluids, hot baths, mild diuretics.

℞ Kalii acetatis	ʒss	2
Aq. fœniculi	℥iij	90

M. Sig.: ʒj every hour if necessary.

Icterus Neonatorum Catarrhalis.

The theories promulgated to explain the causation of icterus in the newborn are so numerous, pedantic and contradicting, that, for the sake of clearness, are best left alone. It is perfectly safe and sane to look upon this common (in about 80 per cent. of all newly born infants) and harmless phenomenon as an expression of the active physiological changes in the liver to which all the other organs are subjected in the first few days of life. It would seem, however, plausible to assume that analogous to catarrhal jaundice in older children, icterus of the newly born is also a manifestation of gastrointestinal irritation, produced by the sudden demand upon the digestive system to exercise functions hitherto not accustomed to.

Hyperactivity of liver.

Gastro-enteric irritation.

The yellowish discoloration of the skin usually appears on the second or third day, first on the face and chest and gradually extends to the abdomen and extremities and, rarely, also to the scleræ. The icterus runs an afebrile, uncomplicated course of about two weeks' duration. Cases proceeding a more protracted course and presenting more or less severe general symptoms should always be looked upon as a partial manifestation of sepsis neonatorum (*q.v.*).

Afebrile course.

Mastitis Neonatorum.

Moderate swelling of the mammary glands of the newborn and discharge of a milklike secretion ("witches' milk") is physiological in infants of both sexes. It begins between the first and third weeks of life and may persist for weeks without giving rise to ill effects. Occasionally, however, as a result of traumatism or infection it may terminate in acute inflammation or even suppuration. In this event the breasts are red, swollen and painful, and may present fluctuation at one or more points, and constitutional symptoms, such as restlessness, vomiting and fever.

Spontaneous.

Traumatism or infection.

If the mammary glands are from the beginning not subjected to meddlesome interference, in short, are left entirely alone, there is usually spontaneous, gradual restitutio ad integrum. Should inflammation ensue, the breasts should be wrapped in oiled cloths or absorbent cotton lightly painted with tincture of iodin, or covered with emplastrum belladonnæ smeared on soft thin leather. In the event of suppuration, if not relieved by spontaneous evacuation of the pus, a radiate incision under aseptic precautions is indispensable.

Evacuation of pus.

Phlegmonous inflammation and gangrene are rare complications, while atrophy of the mammary glands and more or less loss of function may prove very serious to girls.

CHAPTER VI.

Diseases of the Alimentary Tract.

DISEASES OF THE MOUTH.

STOMATITIS.

THIS inflammation of the mucous membrane of the oral cavity is a more or less contagious affection peculiar to infancy and early childhood. It varies in intensity from simple temporary catarrh to fatal gangrene. It is invariably of parasitic origin. The degree of severity of the disease depends upon the pathogenicity of the parasite, the power of resistance of the patient, and the promptness and accuracy of the treatment.

Contagious.

Stomatitis occurs, therefore, principally at a time when the child's health is undermined, as, for example, during dentition, or synchronously with acute infectious diseases. Even normally the mouth forms a favorable nidus for cocci, bacilli, spirilla, leptothrix, and like vegetations, and their growth is surely enhanced by allowing the child to enjoy its acrid nasal discharge; to suck on dirty nipples, toys, and eatables; by keeping its mouth and teeth filthy; by denuding the oral mucous membrane of its epithelium by brisk rubbing in the act of cleansing, and by permitting every friend or kin to infect the child's mouth by overindulgence in the art of osculation. Finally, dental caries, hemorrhagic affections, intoxication from the use of mercury, bismuth, etc., among many other diseased conditions frequently form contributing causes of stomatitis.

Lack of cleanliness.

Local trauma.

Dental caries.

In accordance with the seat and appearance of the lesion it is customary to distinguish the following varieties of the disease:—

1. **Stomatitis Catarrhalis (Erythematosa).**—Redness and slight tumefaction of several portions of the mucous membrane of the mouth, coated tongue with prominent papillæ and red tip and edges. Often marked salivation.

Redness and slight swelling.

2. **Stomatitis Mycotica (Soor, Thrush, Sprue).**—Probably due to a hyphomycete, the *Monilia candida.* Usually begins with a fine, white, flour- or casein-like deposit upon

Flour-like deposit.

Extension to pharynx, etc.

the slightly reddened tongue and buccal mucous membrane. If not arrested the dots and maculæ coalesce and often extend to the pharynx, esophagus, stomach and intestines. This is apt to occur especially in atrophic children.

3. **Stomatitis Maculofibrinosa (Aphthosa, Follicularis, Herpetiformis).**—The causal micro-organism is still undetermined. Often begins with small vesicles. The inflamed mucous membrane is here and there (usually anterior part of mouth) covered by small, grain- to lentil-sized, variously shaped, yellow, grayish-yellow, or grayish-white foci surrounded by a dark-red areola. By coalescence of several follicles large raised plaques are sometimes observed. Fœtor ex ore.

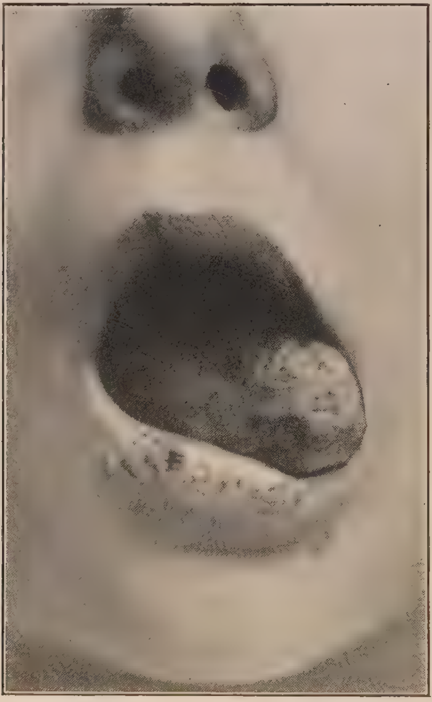

Fig. 52.—Ulcerative Stomatitis. (*Sheffield.*)

Yellowish-gray or white specks.

4. **Stomatitis Ulcerosa (Stomacace).**—It is attributed to the *Bacillus fusiformis* and the *Spirochœte denticola.* The lesion consists of numerous, grayish, irregular ulcers with

may accompany [illegible]

a bleeding base and angry-looking areola, situated at first on the red, spongy and painful gums, and, if not arrested, spreading to the tongue, cheeks or lips. Septic odor ex ore.

Grayish ulcers upon red base.

This form of stomatitis differs from the yellowish to greenish, superficial, easily bleeding ulcers, known as *Bednar's Aphthæ* (ulcera pterygoidea), by the fact that the latter appear symmetrically on each side of the median raphé near the junction of the hard and soft palates.

Differentiation from Bednar's aphthæ and "epithelial pearls."

It may occasionally also be mistaken for the exceptionally ulcerating, so-called *"epithelial pearls."* These innocent milia-like dots, however, are usually found only in the newly born, and situated along both sides of the raphé of the palate.

5. **Stomatitis Gangrænosa (Noma Faciei, Cancrum Oris).**—It occurs principally in cachectic children, chiefly between two and five years old. May follow ulcerative stomatitis or acute exanthematous diseases (measles!). Begins with a small, rapidly spreading, brownish, greenish ulcer upon a hard, elevated base, on the inner surface of the cheek, near the angle of the mouth or on the lips. Very soon a black spot appears on the outside of the cheek, surrounded by marked tumefaction of that side of the face and of the submaxillary glands. The cheek becomes perforated, the edges of the wound turn black, and the sloughing process spreads rapidly so that the whole thickness of the cheek has the appearance of a dirty, greasy scab, and within a few days may be completely destroyed. Sepsis. Rapid exhaustion.

Black spot outside of cheek.

Perforation.

General sepsis.

Mild or even moderately severe cases of stomatitis rarely give rise to systemic disturbance, and unless the local lesion is situated on the lips, tongue, or gums and interferes with sucking or chewing, several days may pass before the disease is detected. Sometimes the patient is feverish and restless, cries and refuses food in the earliest stage of stomatitis, but the constitutional symptoms do not stand in direct ratio to the extent and gravity of the local manifestations. Indeed, the reverse is often the case. However, with persistence of the local symptoms, sooner or later the general health participates in the pathologic process. Starch digestion is greatly impaired by the excessive loss of saliva, which

Mild at onset, hence often overlooked.

almost incessantly dribbles from the swollen, reddened, half-closed mouth, and vomiting and severe diarrhea are frequent results of swallowing of the putrid saliva and the decomposing, more or less ichorous and membranous oral contents. These latter symptoms, in addition to the emaciation from refusal of food and absorption of septic material, greatly delay convalescence and may lead to gradual or rapid exhaustion and fatal issue. In the absence of such grave symptoms and with early and careful treatment the prognosis is good in all forms of stomatitis, except noma (85 per cent. mortality).

Involvement of alimentary tract.

in, Sanious fluid -

Above all, cleanliness should be enforced and the sooner it is begun with the surer we are of rendering the disease free from untoward consequences. Strictest cleanliness of the food, feeding-bottles and nipples, cups, spoons and everything else coming in contact with the child's mouth should be observed. The child's mouth should be regularly washed after each feeding, by *gently wiping* it with absorbent cotton dipped in a 2 per cent. watery solution of boric acid or bicarbonate of soda. As to general cleanliness see "Hygiene" (page 82).

Gentle cleansing of mouth.

In mild cases it is usually sufficient to paint the affected parts once a day with a 2 per cent. solution of nitrate of silver and to employ the following mouth-wash every two to four hours:—

℞ Boric acid,		
Borate of soda	āā ʒj	4
Hydrogen dioxid,		
Glycerin	āā ℥j	30
Alcohol	ʒiv	15
Rose-water	q. s. ℥iv	120

M. Sig.: To be diluted with an equal quantity of water, as a mouth-wash.

Should the stomatitis fail to yield to the treatment after twenty-four or forty-eight hours, more energetic measures should then be adopted to stay its destructive tendencies. The strength of the silver solution should be doubled, and the mouth irrigated every two hours with 1 per cent. permanganate of potash; 5 per cent. Labarraque's solution, etc.

Nitrate of silver.

It is often advantageous to suspend milk feeding for a few days and nourish the child on broths, light cocoa, cereals, toast and tea, pineapple juice, etc. Protracted illness demands active stimulation by means of good wines (diluted), strychnine, and compound tincture of cinchona. This may be combined with the rhubarb and soda mixture, to remedy gastrointestinal disturbance

Stimulation.

Stomachics.

which is ever present in cases of long standing. In the majority of instances even severe cases of stomatitis promptly respond to this mode of treatment. An exception to this rule is made, however, by *noma*,—that rapidly advancing form of necrosis, which knows no barrier to its destructive, death-dealing trail, and often even the knife cannot stay its ravages. At the earliest possible moment the gangrenous portion should be destroyed with the caustic stick, nitric acid or preferably the actual cautery. Frequent cleansing of the parts should be continued day and night, and strengthening food and stimulants administered at short intervals. As Löffler's bacilli were found in a few cases of noma faciei and vulvæ, diphtheria antitoxin (5 to 10,000 units) should be resorted to early in the course of the disease. Very often everything fails; fatal issue occurs either after two or three weeks (sometimes when the patient is apparently saved) or, more rarely, suddenly as a result of entrance of air into the veins. Radical operation has recently received enthusiastic advocacy.

Fatality of noma.

Diphtheria antitoxin.

DENTITIO DIFFICILIS
(Difficult Teething).

As a rule, normal children get their teeth without any difficulty. They may show a slight indisposition in the form of fretfulness, disturbed sleep and slight loss of appetite. If care is being taken not to overfeed the baby during this teething period and the mouth is kept free from outside infection, there is rarely any need for special therapeutic measures. On the other hand, infants of low vitality and more especially those who had been suffering from gastroenteric disturbances or rachitis previous to the eruption of a tooth, teething, particularly when several teeth come at once, is very apt greatly to aggravate the diseased conditions. But, even in these children, neglect in the general care of their health to a great extent is responsible for the serious consequences. Most people are so strongly imbued with the idea that teething is the sole cause of gastroenteritis, bronchitis, otitis and what not, and must be so as a matter of course, that they complacently wait and watch for the teeth to protrude and seek no medical aid to stay the ravages of the incidental ailments. It is usually in these cases that hyperpyrexia and convulsions are encountered, and that remedial measures have to be resorted to, as it were, to facilitate teething.

Slight indisposition.

Not the cause of all ills.

Of course there are infants (see "Spasmophilia") who will get convulsions, high fever, etc., on the most trifling provocation, teething also contributing its share in this direction, but all these extraordinary manifestations are surely exceptional.

Careful feeding.

The main thing, therefore, is to reduce the food, avoid "soothing syrups," which almost invariably contain opium and upset digestion, and to keep the child outdoors.

Where the gum is very much swollen and the tooth is visible under the mucous membrane, lancing of the gum can do no harm and may hasten eruption of the tooth.

DISEASES OF THE SALIVARY GLANDS.

SALIVATION.

Stomatitis.

Prophylaxis.

Increased salivary secretion is almost physiological during first dentition, and is the result of increased blood-supply to the oral mucous membrane. Pathologically it is observed in stomatitis, cretinism, helminthiasis and mercurial intoxication. Occasionally it is met in apparently healthy children long after first dentition, and in the absence of any discernible cause it is attributed to a neurosis. In view of the harmlessness of the condition *per se* no special treatment is indicated except protection of the chin and chest against the irritating effect of the constantly dribbling saliva, and removal of the causes wherever found.

RANULA.

Obstructed salivary ducts.

Retention cysts, congenital or acquired, are not rarely observed in children, and are the result of obstruction of the salivary ducts. Most frequently a globular, usually unilateral, tense, cystic swelling is found on the floor of the oral cavity, sometimes close to the frenulum. This tumor, which is designated *ranula,* varies in size from a pea to a pigeon's egg and contains a thin or viscid fluid. If large in size, the tumor interferes with suckling, swallowing and breathing, and calls for its incision and cauterization, or complete excision.

Sublingual growth.

Ranula is not to be confounded with the peculiar *sublingual growth* (Riga's or Fede's disease) quite frequently observed in Italy[1] among nurslings. This neoplasm is usually situated at the insertion of the frenum linguæ, attains the size of about a five-cent piece and shows a tendency to return unless completely extirpated.

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[1] Only a few such cases have thus far been observed in this country.

SECONDARY PAROTITIS.

This form of inflammation of the parotid gland may occur in connection with acute infectious diseases. It differs from epidemic mumps (*q. v.*) inasmuch as it is, as a rule, unilateral, heals spontaneously within a few days, or ends in suppuration, in the latter event requiring operative interference.

Tendency to suppuration.

DISEASES OF THE TONGUE.

GLOSSITIS.

Aside from the divers pathologic conditions of the tongue ordinarily met in connection with stomatitis, tonsillitis, pharyngitis, exanthematous affections, etc., the tongue is subject to the following peculiar diseases:—

1. Glossitis Marginalis Erythematosa.

The inflammation is usually limited to the edges of the tongue which are red and partially denuded of epithelium. It is observed in artificially fed infants and probably the result of mechanical irritation in the act of sucking.

The treatment is the same as for mild stomatitis.

2. Glossitis Areata Exfoliativa (Annulus Migrans, Lingua Geographica).

As a rule, it begins with a brownish thickening at the margin of the tongue and by gradual spreading forms irregular, circumscribed lines, resembling, as the name indicates, a geographical map. Now and then part of the thickened epithelium is thrust off, but new places are soon involved, and in this manner the affection may go on for years, without, however, giving rise to ulceration of the tongue or any constitutional symptoms. It is not, as was frequently supposed, a sign of syphilis.

Denudation of epithelium.

Not syphilitic.

The treatment consists of cleanliness and occasional painting with a strong solution of chromic acid. (See also Stomatitis.)

DISEASES OF THE ESOPHAGUS.

ESOPHAGITIS.

Primary inflammation of the esophagus is comparatively rare in children, since the principal cause of the disease in the adult, *i.e.*, corroding of the esophagus by caustic poisons taken with suicidal intent, is of exceptional occurrence. However, it is occasionally met in connection with accidental injuries, such as impaction of foreign bodies, unintentional swallowing of caustics, etc., or scalding by hot fluids. The accompanying symptoms vary with the extent of the injury. They consist chiefly of dysphagia, tendency to vomit, and expectoration of bloody, membranous masses. In severe cases, if the patient at all survives (frequently fatal collapse) from the immediate effects of the injury, the esophagitis runs a very protracted course and produces secondary esophageal strictures (*q. v.*).

Accidental injuries.

Dysphagia.

Stricture.

Secondary esophagitis occurs as an extension of inflammatory, especially diphtheritic, processes of the mucous membrane of the mouth and pharynx.

Antidotes.

Anodynes.

Antidotes in cases due to corrosives, morphine hypodermatically for the relief of pain and shock, ice collar to the neck and ice by mouth to subdue the inflammation, and stimulants whenever indicated.

STENOSIS ŒSOPHAGI.

Esophageal strictures may be congenital (*q. v.*) or acquired, the latter being the result of esophagitis (*q. v.*). Depending upon the severity of the injury the stricture may advance up to total atresia. In children the stenosis is most frequently situated in the upper third of the esophagus, and may occasionally be detected by esophagoscopy. Otherwise the diagnosis is established by introduction into the esophagus of an elastic catheter or whalebone provided with small, olive-shaped steel tip. For this purpose the patient is placed in a sitting posture with the head extended slightly backward. The oiled instrument is guided with the first two fingers over the dorsum linguæ and the epiglottis into the esophagus.

Upper third of esophagus.

In acquired stenosis the symptoms usually appear about two weeks after the injury, and consist chiefly of difficult deglutition and gradual loss of weight. In cases of stenosis due to compression of the esophagus by diseased neighboring organs or tumors the symptoms are, of course, more gradual in their development, and more intricate in nature, agreeing with the primary cause.

Dysphagia; loss in weight.

Partial stenoses often yield to dilatation by means of bougies, provided it is continued two or three times a week for at least six months. The bougie is left in place for from five to thirty minutes. Occasional introduction of the bougie after apparent

Dilatation by bougies.

cure will prevent recurrences. Great care and patience are required to prevent perforation. Gavage and nutrient enemata, if necessary. In severe and recurrent strictures operative interference (esophagotomy or gastrotomy). Good results are claimed from the use of thiosinamin. Five drops of a 20 per cent. solution may be injected hypodermatically twice a week, in addition to the dilatation previously spoken of.

Operation.

Thiosinamin.

DISEASES OF THE STOMACH AND INTESTINES.

General Remarks.

Abuse of infantile stomach.

The stomach is the most abused organ of the infantile body, Intended to serve as a recipient of only a sufficient quantity of food to supply the needs of the human organism for its repair, maintenance and growth; destined by means of its juice and ferments to subdivide, assort and predigest the food consumed,—in short to prepare it for easy assimilation; and finally, created to macerate, filter and propel its contents into the channels best suited to complete wholesome metabolism; this very same stomach, regardless of its inherent powers, capacity, state of health, and actual size, only too often is filled to overflowing, forced to "churn" almost incessantly, and to propel the food into the duodenum, frequently long before it is ready for reception. Nay, this very same stomach is rendered a dumping place, during meals, for everything on and off the table, and between meals, for "just a taste" of over- or under-ripe fruit, anilin-dyed sweets and cakes, in addition to the bottle- or breast-feedings given merely as a "drink" to quench the child's thirst. What wonder then that gastroenteric disease is fiercely rampant, that the death-rate from intestinal affections exceeds that of all other infantile diseases combined, and that those unfortunate, foully-fed children who survive remain dyspeptic, rachitic and decrepit, forming an easy prey to acute contagious and infectious diseases that usually befall the faint and the frail! Verily, considering the baleful acts of omission and commission in infant feeding, one is amazed by the ever-swelling hordes of youthful humanity that have apparently escaped the clutches of ignorance —and death. Merciful Nature!

Dumping place for indigestible food.

With the recent advances in bacteriology and physiological chemistry and corresponding improvements in sanitation and infant-feeding, cows' milk no longer holds the record of

"Wuerg-Engel" (destroying angel) of the poor innocent babes. Indeed, seldom a case of gastroenteritis is met which is not primarily traceable to some gross error of diet entirely independent of the cows' milk feeding. The sooner the physician will appreciate that fresh, unpolluted, properly modified (as to quality and quantity), well kept, and regularly administered cows' milk is not inimical (excepted are of course the comparatively rare cases of so-called "cows' milk idiosyncrasy" from birth) to good health and perfect development of the child, the better will he be prepared to reveal the etiologic factors of the gastrointestinal disturbance and combat them!

Clean milk not responsible for disease.

On the other hand, cows' milk, especially in the hot season of the year, whether contaminated at the dairy or at the filthy shop of the remorseless vendor, like water, may form an excellent vehicle for dissemination of pathogenic bacteria, and for the transference of infectious gastroenteric affections.

Whatever the vehicle of transmission,—be it decomposed milk, fruit, vegetables, or meats; infected water, feeding-bottles or nipples, cups or spoons, toys or fingers; infectious discharges from the mouth or nasopharynx, etc.—careful investigation has established the fact that most, if not all, acute gastrointestinal diseases are primarily or secondarily due to microbic invasion of the alimentary canal, the severity of the affection more or less corresponding to the pathogenicity of the invading microorganisms.

Danger in infected milk and other articles of food.

The bacteria responsible for the production of gastrointestinal diseases are very numerous. Streptococci, the bacillus coli communis; the dysentery bacilli of Shiga, Kruse and Flexner; staphylococci, influenza bacilli, the bacillus pyocyaneus, and proteus, among many others, contribute their share as etiologic factors. The determination of the specific germ of each type of gastrointestinal diseases, however, is still a matter of experimental research and subject to great diversity of opinion.

Gastroenteric disorders in breast-fed babies may occur, in addition to errors of diet and exposure to infection—less frequent causes than in hand-fed babies—as a result of disturbance of the quality of the breast milk by disease, fright, grief, privation, pregnancy, and like influences on part of the mother, or wet nurse.

Errors of diet in breast-fed infants.

Finally, even in most carefully fed infants, gastrointestinal disorders are accasionally encountered where the alimentary canal is functionally or anatomically defective from birth or where the infant is suffering from diseases of the other organs of the body, or is indisposed from the effects of functional or organic alterations associated with normal bodily development (*e.g.*, dentitio difficilis!).

Congenital incapacity.

Jmay X

GASTROENTEROCOLITIS

(Dyspepsia, Cholera Infantum, Summer Complaint).

The more critically one analyzes the etiologic factors and pathologic data of the common gastroenteric diseases of early childhood, the more threadbare and misleading appear the existing "text-book" classifications of these affections. It is to be regretted that modern authors still tenaciously cling to and eloquently dilate upon the subdivisions of "gastritis," "enteritis," "colitis," "gastroenteritis," "enterocolitis," and what not, claiming separate and independent existence for each and every one of them, whereas, in reality, neither the clinical signs of a typical case nor the post-mortem findings warrant such an assumption. On the contrary, one is often amazed by the poignant incongruity that prevails between the scarcity and mildness of the post-mortem findings and the extreme gravity of the intra-vitam manifestations of gastroenteric disease in early childhood, and *vice versâ*.

Misleading classification.

Incongruity between intra-vitam and post-mortem findings.

We are inclined to look upon the aforementioned group of gastrointestinal disorders as mere stages of one and the same pathologic condition, and will endeavor to discuss the subject in question from this point of view.

1. Acute Gastroenterocolitis.

Occasional vomiting and diarrhea, occurring as a result of unusual overloading of the stomach, too hasty feeding, partaking of indigestible articles of food (peels and parings) or foreign bodies, exposure to sudden atmospheric changes and undue excitement, etc., are not rarely observed in otherwise apparently healthy, well-nourished children, and if of brief duration, are of no special clinical significance. These attacks may even be accompanied by fever, mild cerebral irritation, colic, etc., and yet remain outside the domain of pathology, or represent an affection which is generally spoken of as simple indigestion or the *first stage* of gastroenterocolitis. By avoiding further transgressions of the ordinary dietary and hygienic rules, and by

Indigestion—first stage.

removing the causal obnoxious influences recovery is usually prompt and permanent.

If, however, the vomiting and diarrhea persist or recur at frequent intervals; if the child loses its appetite and some of its weight; if its tongue becomes heavily coated, its abdomen greatly distended and its general health more or less seriously impaired; if the infant suffers from severe abdominal pain after each feeding and vomits part of the food consumed and some mucus and bile; finally, if the stools rapidly increase in number and consist of masses of undigested food, of bad color and offensive odor, a symptom-complex develops which represents the *second stage* of gastroenterocolitis and is generally described as gastrointestinal catarrh or dyspepsia.

Dyspepsia—second stage.

Ordinarily these manifestations set in insidiously and if not promptly arrested grow worse gradually, arousing little if any anxiety on the part of those in charge of the baby, or are lost sight of, sometimes because of coincident "teething" (with the laity the presumptive cause of all ills), until there is a sudden aggravation of the condition—supervention of the *third stage* of the disease.

Gastroenterocolitis—third stage.

In this stage gastroenterocolitis assumes a very acute course. It is manifested by violent vomiting, excessive thirst; frequent, thin, watery, brownish, greenish, and later colorless or blood-stained stools. The vomitus is acid in reaction, bile-stained, and offensive in odor. The bowel movements vary between ten to fifteen in twenty-four hours, are preceded and followed by griping pain and tenesmus. The child is very restless, feverish, sleepless, and, with the symptoms persisting a few days, rapidly loses in weight, and sinks into a state of collapse, followed by convulsions, coma and death. More favorable cases may improve under energetic treatment, or linger for weeks or months, frequently suffer from intense exacerbations of the attack, and, finally, either recover after tedious convalescence or die from inanition or complications.

Closely allied to the gastroenterocolitis just described (though possibly differing as to the exciting micro-organism), and probably representing only a severer, "fulminating" form of the same disease, is the so-called infantile "summer-complaint" or *cholera nostras s. infantum.* It usually rages in epidemic form during the hot summer months, especially among bottle-fed infants and those exposed to bad hygienic conditions, but occurs sporadically

Fulminant form.

Often epidemic.

also at other seasons of the year. As with other contagious and infectious diseases previous ill health serves as an active and favorable predisposing cause also in this destructive affection, the acute and grave symptoms ordinarily supervening upon a latent period of indisposition of variable duration..

Collapse.

The attack ushers in suddenly with vomiting, diarrhea and prostration. The vomiting is more or less projectile in character and occurs especially immediately after drinking. The evacuations range between fifteen to thirty or more in twenty-four hours, are at first fecal in consistency and odor, but soon turn very watery, serous, light yellow or greenish in color, and occasionally mixed with blood-streaked mucus. The abdomen is often trough-shaped and but slightly sensitive to pressure. The thirst is intense; the tongue dry, brown or black and cracked, irrespective of the degree of temperature, which is rarely very high. Owing to the excessive loss of fluids the urine is very scanty and often contains a moderate amount of albumin.

Watery, mucous and blood-streaked stools.

As the disease progresses the child perceptibly loses in weight, from hour to hour; its face is pinched; its fontanelles, temples and eyes are deeply sunken; its extremities are cool and blue; the heart-beat and respiration barely audible—in short it is in a state of profound collapse. Apathy, somnolence, convulsions and death then follow in rapid succession; the younger the child, the earlier, as a rule, the fatal termination. The latter is sometimes preceded by a state of *hydrocephaloid*—a condition variously ascribed to cerebral anemia or hyperemia, edema of the meninges and uremia, and presenting the following symptom-complex: *First stage*, fever, restlessness, jactitations, and insomnia, flushed face, strong and bounding pulse; *second stage*, subnormal temperature, cold extremities, feeble, irregular pulse and respiration, apathy, sopor and coma.

Intense emaciation.

Hydrocephaloid.

The disease having reached this grave stage it offers a very bad prognosis; but few children manage to survive so violent an attack. Some of the few who do are apt to succumb later to complicating nephritis, pneumonia, cerebral sinus thrombosis, peritonitis and the like.

High mortality.

Convalescence is very tedious even in the absence of complications, and a great many children remain decrepit for life, chronic otitis media, xerosis of the cornea and panophthalmia often adding their share of misery.

With such sad prospects in view after the gastrointestinal affection is fully established, the urgency of early and energetic prophylaxis and treatment can readily be appreciated.

Preventive measures.

Sterilization of milk.

Change of air.

Breast feeding.

To prevent the graver forms of gastroenterocolitis we must promptly remove the causes and effects of the mildest symptoms of the disease. Attention to every detail of rational feeding and personal hygiene and strictest cleanliness of the child's living rooms, feeding utensils, wearing apparel, and of all other things coming in direct contact with the patient are the surest means of prevention. As in the majority of instances the pathogenic bacteria enter the infantile alimentary tract with infected milk or water, these should, especially in the summer months, be sterilized or even boiled, regardless of the temporary arrest of gain in weight that is concomitant with such feeding—a puny baby on the lap rather than a fat one in the grave! Weaning of the baby and other innovations during the hot summer months should be avoided. Lengthy voyages exacting prolonged disturbance of rest, sleep, and proper feeding should be interdicted. On the other hand, a sojourn in the country (inland, mountains, or seashore) should be encouraged. Last but not least in importance as a prophylactic measure is the practice of whole or partial breast feeding of infants under one year of age, unless countermanded by definite contraindications.

Discontinuance of milk.

The active treatment should begin, as already suggested, with the earliest inception of the gastrointestinal disorder. Regulation of diet is our most efficient therapeutic measure, and is almost invariably attended by improvement in the child's condition if it is begun with a few hours' starvation of the patient and prompt cleansing of the alimentary tract of its obnoxious contents. Feeding, breast or bottle, should at once be suspended until such time as exigencies for resumption of feeding will demand. In the mean time, especially in the absence of strong contraindications, such as violent vomiting, the infant should receive small quantities of hot or cold pure water or a light infusion of black tea. Recurrent vomiting calls for prompt attention especially because of its fearfully exhausting effects, but also because it greatly hinders in the administration of suitable medication. Ordinarily vomiting can be controlled by "ice-sand," minute doses of calomel with or without bicarbonate of soda or bismuth; bismuth and cerium oxalate; tincture of iodine (in $\frac{1}{30}$ of a drop doses, to be repeated every hour or two); silver nitrate (gr. $\frac{1}{100}$); a sinapism to the spine or epigastrium, and if all else fail, lavage. In hospital practice the order of these therapeutic suggestions is usually reversed, *i.e.,* lavage is usually resorted to first, and as a rule with immediate relief to the patient. In private practice, however, one often meets with objections on the part of parents, and hence is obliged to primarily "medicate.") Lavage should be supplemented by enteroclysis and, with the vomiting checked, also by a small dose of castor-oil.

Lavage and enteroclysis.

This mode of treatment generally suffices to arrest gastrointestinal affections of moderate severity. Where the diarrhea persists we are often called upon to administer an astringent mixture like the following:—

Astringents.

℞ Bismuthi subcarbonatis,
Mist. cretæ comp.,
Syr. rhei aromat.,
Glycerin.,
Aq. menthæ pip. ....āā ʒij | 8
Aq. destil. ....q. s. ad f℥ij | 60
M. Sig.: One teaspoonful every two hours for a child one year old.

The camphorated tincture of opium may be added for the relief of pain. After complete cessation of vomiting, we may resume feeding, first with small quantities of toast- or barley-water and, several hours later, diluted, sterilized cows' milk or breast milk.

Cereals.

In fulminating attacks of gastroenterocolitis where the bacterial toxins so violently overwhelm the infantile organism, producing intense shock, the treatment must be very prompt and more heroic. In the *initial,* febrile stage, after a single but thorough irrigation of the stomach and bowels the little patient is given one-fiftieth of a grain of morphine and one five-hundredth of atropine hypodermically, is wrapped in warm blankets and sent outdoors—wherever a good breath of air is obtainable—preferably to the seashore. After responding favorably the treatment is followed up in the manner previously outlined for less severe cases.

Morphine and atropine.

In the *algid* stage, where the child is at death's door—wasted, cold, blue, rigid and lifeless, in short in profound collapse—powerful stimulation is in order. Thus, a hot bath with brisk rubbing of the body; a hot, high enema (injected slowly so as to be retained), hot water by mouth, hypodermatic administration of sterile camphorated oil (8 drops of a 15 per cent. solution), strychnine (gr. 1/60 to 1/30), caffein sodium benzoate (gr. j), or whiskey (gtt. x), and hypodermoclysis (1 to 6 ounces of a

Heat.

Stimulation.

0.6 per cent. hot sterile salt solution). As the patient improves a milder course of treatment is, of course, resorted to. The physician should not be deceived, however, by those apparent improvements, as they not rarely precede fatal termination.

[illegible] – [illegible] Colitis [illegible] 233 –

2. Subacute and Chronic Gastroenterocolitis.

Delayed convalescence.

Recurrences.

Exhausted by the paralyzing action of the virulent bacterial toxins; wasted and weakened from the excessive loss of body fluids and the strict starvation diet enforced during the acute course of the disease, the little patient rarely, if ever, emerges in a state of health capable to exercise its digestive organs to their normal capacity. On the contrary, convalescence usually proceeds at a very slow pace, and is frequently interrupted by milder exhibitions of gastrointestinal indigestion which, if not promptly yielding to energetic treatment, eventually lead to chronic involvement of the alimentary tract.

Pathologic findings.

The mucosa of the stomach and bowels, especially of the ileum and colon, undergoes gradual thickening, ecchymosis and ulceration. The mesenteric glands are more or less enlarged, and on cross-section partly red and partly yellowish gray in color and sometimes caseated. In very protracted cases the mucosa and its follicles are atrophied, and the lungs, liver and heart in a state of inflammation and degeneration.

Diarrhea.

Persistent loss in weight.

The bowel movements continue to be frequent (four or five times in twenty-four hours). The stools are thinner than normally, are mixed with particles of undigested food, mucus, and blood. The abdomen is flat, sometimes deeply sunken, and through its thin and wasted wall one can readily palpate the greatly enlarged, "ropy," mesenteric glands. The child's appetite is capricious, often rather very good, and contrasts strongly with the persistent loss of weight. The tongue is coated and flabby, its edges are red and indented by the teeth or gums, and here and there covered by an aphthous deposit. Slight indiscretions in the dietary are promptly followed by vomiting and diarrhea. Chemical examination of the contents of the stomach discloses marked diminution of hydrochloric acid.

The course of chronic gastroenterocolitis varies in individual cases. Some infants, especially those in whom the chronic affection followed upon the acute form, who remained free from grave complications and retained some vitality, often unexpectedly show marked improvement with the setting in of cooler weather, and regain their health fully within but a few weeks.

Variable prognosis.

In another group of cases recovery is less rapid. Improvement alternates with aggravation of the condition, but, finally, the infant extricates itself barely alive, with a load of sequelæ (*e.g.*, rachitis) which keep it in a state of decrepitude for many years after, and not rarely for life.

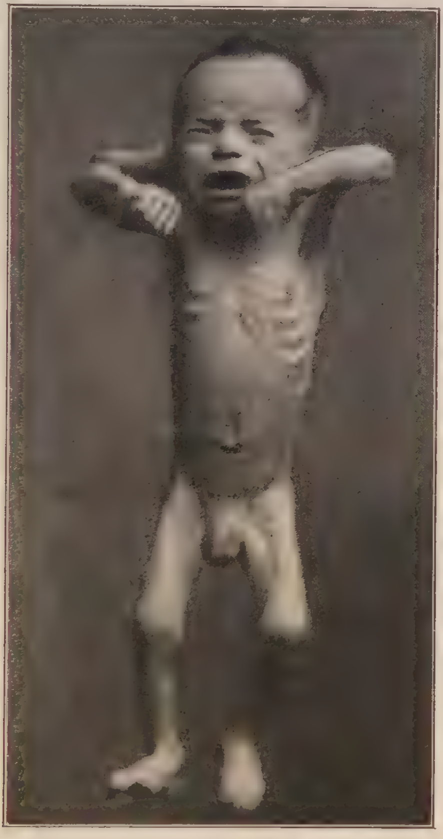

Fig. 53.—Gastroenterocolitis Chronica in a child 10 weeks old. Suffering also from Tetanism. (*Sheffield.*)

Grave symptoms.

In still another group of cases all therapeutic efforts utterly fail to effect a cure. The child's face has a pallid, earthy tint, and senile expression; the skin is dry and hangs in folds; the fontanelles and temples are depressed, and after a period of several weeks or months the infant finally succumbs either slowly with symptoms of cerebral anemia and heart-failure or suddenly during an attack of eclampsia. The fatal termination is frequently enhanced by complicating pulmonary (passive- or broncho-pneumonia) and renal (colicystitis, pyelitis, etc.) affections; skin (ecthyma, furunculosis), ear and mouth infections, or intercurrent acute communicable diseases (exanthemata).

Complications.

At best the prognosis is very grave (30 per cent. mortality), especially so in infants reared under bad hygienic conditions, in want and misery, and in those born with lowered vitality and congenital defects.

However, no effort should be spared to save an infant that is apparently hopelessly lost, for just in chronic gastroenterocolitis the unexpected sometimes happens—recovery takes place at a time when relief by death is prayed for.

Unexpected recovery.

The patient should be removed from unsanitary surroundings and intrusted to the care of someone who would *obey* orders and not use her own judgment and that of the many "good and experienced" neighbors. Be it remembered that only too often change of nurse (with her gross negligence and stubborn interference) has saved many a hapless baby! Regulation of diet is most essential. No hard and fast rule, however, can be laid down in this direction. We must feel our way in every individual case. It is always a good plan in bottle-fed babies to begin treatment with discontinuance of the milk for a day or two and thorough cleansing of the alimentary tract by lavage and enteroclysis. In the mean time the patient should be fed on thin barley-water, a light infusion of black tea, albumin-water, and, perhaps, a small quantity of freshly boiled, fat-free chicken soup. As soon as the stools diminish in frequency and improve in consistency, we resume milk-feeding in very high dilution. For a child let us say six months old we prescribe one teaspoonful of milk to seven teaspoonfuls of barley- or rice-water, to be given every three hours, and direct daily to increase the quantity of milk until the percentage of one to three has been reached, and then gradually to augment the total quantity at the last ratio (*i.e.*, 1 to 3), until six ounces are obtained for each feeding. Should the milk mixture disagree, a weaker milk mixture is resorted to, or milk is again discontinued, falling back upon cereals, albumin-water and tea. Some infants do well, at least for a time, on condensed milk and barley-water; others, especially those suffering from the so-called "fat-diarrhea," improve rapidly

Change of nurse.

Regulation of diet.

Cereals, later with milk.

Whey in fat diarrhea.

Malt-soup.

on skimmed milk or whey, and still others (older ones), who cannot tolerate milk in any form, get along on toast and tea, cocoa in water, mashed potato with beef juice or chicken soup, soft-boiled egg, custards and similar semisolid articles of food. In a great many instances "malt-soup," prepared in accord with the directions of Keller, acts admirably, both as a tissue builder and to check the protracted diarrhea. Last in line, but foremost in importance, is the fact that a complete cure of chronic gastroenterocolitis in bottle-fed infants is almost invariably effected by a prompt change from bottle- to breast-feeding.

Enteroclysis.

Tannates.

Bismuth.

The medicinal treatment of chronic gastroenterocolitis is chiefly symptomatic. Where vomiting persists, lavage (with warm boric acid solutions) should be practised daily or every alternate day, and continued for a few weeks. Digestion may be aided by means of pancreatin and diastase, and the appetite improved by small doses of tincture nux vomica and cinchona. The patient should be given daily a high intestinal irrigation, either with one quart of plain hot (105° F.) water, 2 per cent. of bicarbonate of soda, or, where the lesion is localized principally in the lower bowel—as indicated by predominance of blood and mucus in the evacuations—with $\frac{1}{10}$ per cent. solution of nitrate of silver. Where the diarrhea persists notwithstanding progressive improvement in the general condition of the patient, the newer tannin preparations (*e.g.*, tannalbin, tannigen) are very serviceable. The tannates may be combined with some bismuth preparation (*e.g.*, subgallate of bismuth gr. ij to iv), to enhance the astringent effects.

Hyg-

Change of air (seashore), strict cleanliness of the body, change of position and frequent picking up of the patient from its bed, and active stimulation (strychnine, cinchona, Tokay wine and champagne) are active preventives of serious complications.

PROCTITIS.

Secondary.

Inflammation of the rectum is usually secondary in character, and not rarely associated with gastroenterocolitis, dysentery, oxyurides, and prolapsus recti, and less frequently with gonorrhea (vulvovaginitis, *q. v.*) and diphtheria. Occasionally it is the result of trauma (*e.g.*, foreign body), and the effect of drastic cathartics.

Tenesmus.

The principal symptoms of this affection consist of tenesmus (sometimes also strangury), frequent discharge of blood, mucus, and pus, with little fecal matter, and more or less severe colic. Depending upon the primary cause of the disease, the discharges may contain different kinds of bacteria (*e.g.*, ameba, gonococcus, diphtheria bacillus, worms, etc.), a fact which should always be borne in mind before arriving at a diagnosis and resorting to treatment. Proctitis should not be confounded with rectal fistula or hemorrhoids, purpura hemorrhagica and intussusception. The treatment depends upon the underlying cause; in the main resembling that of dysentery (*q. v.*).

Differentiation from hemorrhoids, fistula, purpura and intussusception.

COLICA INFANTUM, GASTRALGIA, ENTERALGIA, NEURALGIA ENTERICA.

Infantile colic is usually associated with a number of congenital (gastrointestinal stenoses, etc.) and acquired (gastrointestinal inflammations, etc.) diseases of the alimentary tract. Less frequently it is apparently free from organic underlying causes. This so-called "idiopathic" form of colic is a spasmodic affection of the intestinal musculature, the result of pathologic irritations which act by way of the peripheral cutaneous nerves or the sensory intestinal nerves. To avoid unnecessary repetition, it may briefly be stated that anything capable of producing gastrointestinal disturbance may form the cause also of the said pathologic irritations. This occurs especially in premature infants and in those whose digestive organs are not quite fully developed.

Digestive disturbance.

Functional incapacity.

Some babies, breast- or bottle-fed, begin to suffer from colic soon after birth, and do what you will, maintain their "record" for several months,—until, with gradual growth, the digestive organs attain their normal functions. Such "colic-babies," if reared without immediate strict supervision of a capable nurse or physician are apt very soon to contract a severe gastrointestinal disorder from the effect of the experimental efforts, in feeding and medication, on the part of all who sympathize with the "innocent babe." This *habitual* colic, which is manifested by continued fretfulness, sleeplessness, and *pseudo*bulimia (instinctive, eager desire for warm drinks which temporarily relieve the pain), is to be distinguished from *acute* intestinal colic (colica flatulenta), which is sudden in development and rapid in disappearance, the latter depending upon the time required to get rid of the gas or stool. During a severe attack of acute colic the child's face is spasmodically drawn and bathed in perspiration.

Habitual.

Flatulent.

Spasms.

The patient refuses food, cries pitifully, and draws its legs upon the abdomen. The spasm sometimes extends to the other muscles of the body, leading to general convulsions, and exceptionally even to coma and fatal issue. Of course, in the great majority of instances, the termination is favorable, especially under prompt and appropriate treatment.

Prevention of constipation.

In breast-fed infants attention to the health of the mother or wet-nurse—avoidance of excitement, regulation of the bowels, indulgence in outdoor exercise—and, in both breast- and artificially fed, prevention of constipation and overfeeding of the infant are very efficient anticolic measures. Where repeated examination of the breast milk proves it to be too rich in fat or proteids, the infant should be given a few teaspoonfuls of water or of some other diluent immediately before each nursing, and the length of time for each nursing proportionately reduced.

As long as the infant thrives, notwithstanding the colic, no very material changes in the feeding should be attempted, as too much experimenting often makes matters worse.

Heat externally and internally.

In habitual as well as flatulent colic, heat, either in the form of fomentations (a few drops of turpentine in a quart of warm water), gentle massage of the abdomen with warm oil, or warm drinks such as chamomile-, fennelseed- or peppermint-tea, will be found to act well. In cases of acute colic this must be preceded by a warm-water enema to aid in the expulsion of the gas or stool. Of drugs the following preparations are worth trying:—

Soda and mint.

Charcoal and magnesia, of each 1 or 2 grains one hour after feeding; mistura sodæ et menthæ, N. F., 5 to 10 drops every ten minutes until relieved; compound spirits of ether, sweet spirits of nitre, or camphorated tincture of opium in doses of from 2 to 5 drops, to be repeated two or three times. In purely nervous colic asafetida often acts magically. The lac asafetida (℥ss to Oj of warm water) should be gently administered by rectum. The ammoniated tincture of valaria (gtt. v) and sodium bromide (gr. ij) are often equally efficient. As to the treatment of convulsions see page 554.

Asafetida per rectum.

Proper food, regular bowel movements, and fresh air are efficient prophylactic measures.

Differential diagnosis.

Infantile colic should not be confounded with intestinal intussusception, appendicitis, and biliary, renal (uric acid infarct!) or vesical calculi.

CHRONIC CONSTIPATION.

Sequelæ of constipation.

Judging by the construction of the infantile intestines—their great length, the thinness and feebleness of their musculature, etc.—nature seems to have intended that infants as well as older children should be more or less constipated. Indeed, the popular belief that healthy children are usually constipated is often corroborated by actual observation. Not infrequently, however, obstinate constipation gives rise to a number of disagreeable symptoms (flatulence, anorexia, headache, restlessness, sometimes convulsions, proctitis, anal fissure, prolapse of the rectum, hemorrhoids, etc.) requiring active treatment, a task often difficult to cope with in view of the uncertainty of the etiological factor of the underlying disease.

Congenital malformations.

The causes of habitual constipation are very numerous. Aside from the cases resulting from gross abnormal anatomical relations or diseases, such as the different varieties of atresia intestini, recti, or ani; tumors; congenital dilatation with hypertrophy of the colon; hypertrophy of the valvulæ conniventes; hypertrophy of the so-called rectal valve; inflammatory adhesions; congenital displacements, etc.—which will not be discussed here—constipation is ordinarily caused by faulty diet, atony of the bowels, and constitutional disturbances.

Lack of fat.

Faulty diet is responsible for a great many cases of constipation. This etiological factor is frequently potent also in infants, when the woman's milk contains too much or too little of one or more of the constituents of milk, or is insufficient in quantity. In artificially fed infants the cause of the constipation will probably be found in the insufficiency of fat consumed. In some children constipation is due, on the one hand, to too early and persistent feeding with amylacea, and, on the other, to the consumption of food that does not stimulate peristalsis, such as an exclusive diet of milk, meat, eggs, etc., and no potatoes, bread, fresh vegetables, etc.

Muscular insufficiency.

Atony of the intestines may be primary, congenital in nature, or secondary or acquired. The former variety can frequently be traced as an hereditary taint through several generations. Sometimes there is, in addition to the muscular insufficiency, also congenital weakness of the innervation of the intestines. The latter condition embraces also the form of atony usually associated with congenital diseases of the brain and spinal cord. Secondary or acquired intestinal atony is generally the result of repeated attacks of temporary constipation, gastrointestinal indigestion with fermentation, enterospasm, arrest of peristalsis due to reflex irritation of the inhibitory nerves of the intestines, acute inflammatory processes of the intestinal canal with consecutive atrophy of the intestinal coats, constriction of the lumen of the bowels by temporary displacements (enteroptosis, hernia, etc.), habitual suppression of defecation or attention to it at irregular hours, enemas with large quantities of fluids, etc. All these etiological factors produce intestinal atony by directly or indirectly distending the lumen of the bowels and depriving the intestinal musculature of its resilience and tonicity. The latter condition is also apt to follow the abuse of antispasmodics, while drastic cathartics may lead to atony by mechanically thinning the intestinal coats.

Enterospasm.

Hernia.

In different chronic diseases associated with general debility (*e.g.*, rachitis) and loss of flesh; in diseases of the nervous system, such as locomotor ataxia, myelitis, meningitis, etc., the sluggishness of the bowels forms merely a symptom of the principal disease. Habitual constipation is often met in diseases of the heart, profound anemia, etc., as a result of venous stasis of the abdominal organs; to the same cause is attributable also the constipation occurring in children who through deformity or otherwise are incapacitated to enjoy a sufficient amount of bodily exercise.

Chronic organic diseases.

The *treatment* of obstinate constipation in infancy and childhood resolves itself, firstly, in arresting the causes instrumental in the production of the disease; secondly, in the removal of the damage done during the continuance of the constipation—not quite as easy a task as some authors wish us to believe. Indeed, a good number of cases of chronic constipation are never cured, no matter what therapeutic means are being employed. Preventive measures are, therefore, to be recommended early and carried out with precision.

It is of primary importance to train the child to have a movement regularly every day. Proper habits are often easily formed if the child is put upon the chamber or chair invariably at the same hour. The first few days it may require local stimulation to defecation (*e.g.*, introduction into the rectum of a small oiled syringe-tip). Similar means should be employed also with older children and particularly school-children, who are very apt to suppress nature's impulse to empty the bowels.

Regular habit.

Suitable commode.

Two main factors are instrumental in the expulsion of the rectal contents: Contraction of the abdominal muscles and the diaphragm, and separation or relaxation of the gluteal group of muscles. If the seat of the commode is too high and the aperture in the seat too wide no support is given to the tubera ischii, the gluteal masses are crowded together instead of separated and the descent of the floor of the perineum is much hindered. This impediment to defecation may be obviated by substituting a low seat on a nursery chair or closet or small vessel for the high one previously used. The child is thus enabled to accomplish this act in a squatting posture which is most favorable to thorough emptying of the rectum.

Proper diet.

Increase in fat.

Mixed diet.

Correction of diet is, of course, very valuable for the prevention of habitual constipation, but does not always remedy the trouble. This is particularly true of cases of very long standing, since here we are dealing with secondary atony following prolonged distention and enfeeblement of the intestines. With the introduction of the recent methods of percentage feeding and the employment of "top milk" as a base, and barley- or oatmeal-water as a diluent, the number of cases of obstinate constipation among bottle-fed infants, due solely to faulty feeding, has perceptibly diminished. Hence, the indication of these methods of feeding also as a corrective of constipation. In breast-fed infants attention should be directed to the improvement of the general nutrition of the mother or wet-nurse. Frequently, however, it is almost impossible to regulate the quantity of fat in breast-milk. In this event the deficiency in fat may be supplied by administering to the infant, just before nursing, a teaspoonful or two of sweet cream. The addition of cream, malt preparations, buttermilk, honey, an extra supply of cooked or raw fruit and vegetables to the regular "mixed-diet" is invaluable as a corrective of constipation also in older children. A glass of cold water on an empty stomach and at night before retiring is often very useful.

Faithful compliance with the suggestions just made very often yields favorable results. In a certain percentage of cases, however, more active measures have to be resorted to and it then devolves upon the physician to select such therapeutic means as will not affect the general well-being of the patient. This indication can most appropriately be met by the simultaneous employment of a combination of the so-called physicochemical procedures, consisting of massage, oil enemas and hydrotherapy, and occasionally, also, electricity. This treatment is more advantageously carried out in the evening, before the patient goes to sleep. The child is placed on a hard couch or mattress with head and thorax raised and legs sharply flexed at the knee-joint and somewhat rotated outward. The attendant stands on the left side of the patient. The manipulations are begun at the fossa iliaca sinistra, where the sigmoid flexure is situated and is frequently found to be a halting place for hardened feces. With the tips of the fingers of one hand (in older children both hands may be used, one hand being placed upon the other) the attendant makes gentle circular movements along this portion of the colon and at the same time exerts upon it considerable pressure downward toward the rectum. Without changing these movements the attendant slowly ascends as far as the splenic flexure. From here he gradually returns to the sigmoid. He now begins a new tour going as far as the hepatic flexure, and after gradually returning to the starting point he makes his final trip reaching the cecum and, in the manner just outlined, returns again to the fossa iliaca sinistra. These manipulations should be followed by rhythmical vibratory strokes over the entire abdomen, interrupted by a few pressure movements against the spinal column in the epigastric region. The treatment should last from six to twelve minutes.

Massage.

Instead of trying the massage, oil enemas, and hydrotherapy separately, it is certainly preferable to employ these three procedures—the *anticostive triad*—simultaneously, as they do not interfere with one another, but, on the contrary, are destined to supplement one another in their beneficial effect. Thus, after completing the massage the little patient is turned upon his left side, and by means of a piston syringe half an ounce or more of oil is gently injected into the rectum and allowed to remain there. This is followed by the application around the abdomen of a Priessnitz compress, which should be left in place until the next morning. It will almost invariably be found that the patient's bowels will act either during or soon after the treatment or, at any rate, not later than the following morning. A three or four weeks' course of treatment will usually suffice to establish regularity of the bowels provided the preventive measures suggested before are strictly adhered to. In some, very protracted, cases of constipation these procedures may be supplemented by the application of the galvanic or faradic current. One electrode is passed

Anticostive triad—massage, oil enemas and hydrotherapy.

Persistence in treatment.

successively over different portions of the abdominal wall, and the other electrode is placed upon any other part of the body.

Divulsion in rectal involvement.

Proctologists frequently advocate divulsion of the sphincter ani as a sure cure of habitual constipation. I am not inclined to be quite as enthusiastic over it, except in cases of constipation due to rectal disease, as, for example, fissura ani, rectospasmus, etc.

Laxatives.

Finally, there is a class of cases of chronic constipation which resists all forms of treatment as regards a permanent cure, but may be considerably improved by alternately resorting to the therapeutic measures already enumerated as well as to drugs. In the selection of an evacuant the physician must be guided by the etiological factors and the individual peculiarities of the case in question. The indiscriminate use of antispasmodics as well as the ever-ready, "soothing" laxatives is to be strongly deprecated. Effective and comparatively harmless are the following remedies: Soap and glycerin suppositories, medicated cocoa butter suppositories (with aloin and belladonna in spastic, or aloin and nux vomica in atonic, constipation), enemas with small quantities of glycerin or larger quantities of soap-water; internally magnesia usta, magnesia and rhubarb, compound licorice powder, castor-oil, extract of cascara sagrada, calomel followed by a mild saline aperient, and, in older children, the standard mineral salts or waters.

Suppositories.

Whatever the method of treatment employed, the establishment of a *habit* to move the bowels regularly at a certain time of the day should at all times be our chief aim.

PROLAPSUS ANI, PROLAPSUS RECTI.

Round, red mass.

If the prolapse is limited to the mucous membrane of the anus, the condition is spoken of as prolapsus ani; if the lower portion of the rectum protrudes through the anal orifice, it is known as prolapsus recti. In prolapsus recti the protruding part comes down during defecation in the form of a round, or sausage-shaped, glistening, red or bluish red, frequently bleeding mass. In the beginning the mucous membrane slips back in its place spontaneously, or is easily replaceable and remains there until the next movement; in severe cases, owing to marked inflammatory thickening, reposition of the mass may be very difficult, and if replaced may immediately prolapse again.

These conditions are very common in young children, the softness of the connective tissue and incomplete development of the muscular system serving as predisposing causes. The ordinary exciting causes are habitual constipation, protracted diarrhea, proctitis, rectal polypus, oxyuris, phimosis, vesical calculus, *i.e.*, conditions in which the act of defecation or urination is attended by pressing, tenesmus or strangury. Protracted, paroxysmal coughing (*e.g.*, pertussis), by its downward pressure upon the abdominal contents, also serves as an etiological factor, and prolapsus recti is not infrequently associated with rachitis, probably due to the accompanying muscular debility and constipation.

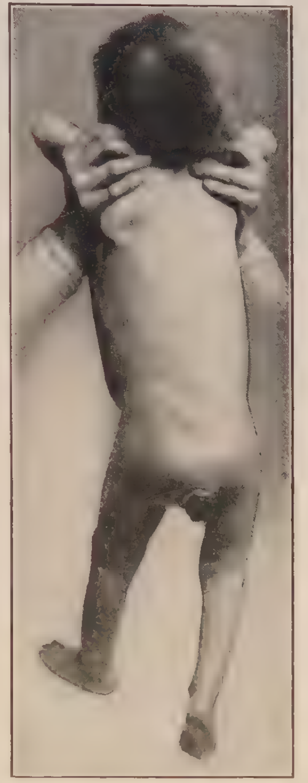

Fig. 54.—Prolapsus Recti. (*Sheffield.*)

Diagnosis by inspection and digital examination.

The diagnosis can readily be made by inspection and digital examination. It is most apt to be confounded with hemorrhoids and rectal polypus. Rectal polypus is the most frequent cause of rectal bleeding in children, and appears at the anus as a dark-red, bean- to cherry-sized, roundish tumor with a bleeding surface. Digital examination usually reveals that the polyp is attached to the rectum, a few centimeters above the sphincter, by means of a short or long pedicle.

Differentiation from hemorrhoids, polypi and intussusception.

Reposition and strapping.

Slight prolapse is readily amenable to reposition of the prolapsed mass (oiling and gentle pressure upward with the patient in knee-chest position) and strapping of the buttocks (in older children only before the act of defecation), in addition to prompt attention to the aforementioned etiologic factors. Severer cases call also for reduction of the local inflammation by occasional painting of the affected area with balsam of Peru or a 2 per cent. solution of nitrate of silver. If these measures fail the prolapsed mass should be treated by punctate or linear cauterization.

Nitrate of silver.

Actual cautery.

General tonic treatment not rarely succeeds where local procedures fail.

INTUSSUSCEPTION
(Intestinal Invagination).

Intussusception, or sliding of one portion of the intestines into the other, is an affection principally of infancy and early childhood. Seat of lesion. The commonest seat of the trouble is the ileocecal region. Thus, the proximal portion of the ileum with or without the cecum becomes invaginated into the colon. Less frequently the ileum slides into the ileum, or a part of the colon into the colon. The immediate results of the invagination are agglutination of the opposed serous layers and strangulation of the impacted portion of the intestine. Strangulation of impacted portion. If the latter is not soon relieved, gangrene, sloughing and spontaneous discharge of the cast-off piece of intestine occurs,—the continuity of the intestine being preserved by end-to-end adhesion.

The disease sets in very suddenly. In the midst of apparently perfect health, the child suddenly shrieks from intense pain and presents other symptoms of severe colic which fail to yield to ordinary anticolic therapeutic measures. Sudden intense pain; tenesmus. The pain and restlessness increase, the abdomen becomes greatly distended, and, accompanied by marked tenesmus, the child passes from the bowels at first small quantities of feces mixed with mucus and blood, and later pure blood.

Digital examination discloses blood in the rectum—often long before any is passed with the stool,—and if the intussusception is colonic in form, frequently a round mass is observed high up in the rectum. Mass in rectum. Exceptionally and late the tumor protrudes from the anus. In ileocecal intussusception inspection and palpation reveal a round "lump" or sausage-shaped mass in the right iliac region, and occasionally a depression below the tumor—owing to displacement of the cecum. The tumor is less pronounced in intussusception of other portions of the intestines.

The severity of the onset is no criterion as to the further course of the disease. In a small number of cases the colic suddenly ceases, the child resumes his normal appearance, and exhausted from the agonizing pain he falls into a profound sleep, waking up apparently well—spontaneous improvement or recovery by spontaneous reduction of the invagination has apparently occurred. Spontaneous recovery. In such a cure the trouble is not always at an end, for the intussusception is very apt to return after a shorter or longer interval. In another group of cases, after the grave onset, the disease may pursue a milder course. The vomiting, meteorism, and tenesmus abate in their violence; the dejecta lose their bloody consistency, and the colicky pain returns only after long pauses. After three or four days, a piece of gangrenous intestine, the intussuscepted portion, is discharged per rectum. This process is always fraught with danger. The few patients who survive frequently succumb to consecutive chronic gastro-intestinal catarrh, with or without intestinal stricture. In the majority of instances, the symptoms grow worse within twenty-four hours from the start of the attack. The vomiting becomes violent and stercoraceous, the pulse feeble, the extremities cold, the expression of the face pinched, the eyes sunken, and unless the condition is promptly relieved, the child succumbs within from four to eight days to increasing collapse, not rarely preceded by intestinal perforation and peritonitis.

Discharge of intussuscepted portion.

Peritonitis; collapse.

At all events the prognosis is very grave. The mortality ranges between from 50 per cent. and 80 per cent. in cases left alone or treated palliatively. On the other hand, with prompt surgical treatment the chances for recovery are by far better—about 65 per cent. The best results (75 per cent.) are obtained in cases operated upon within 24 hours of onset of attack.

Prompt surgical treatment.

The treatment of choice, therefore, is obvious: Early operative interference,—before extensive adhesions and gangrene of the bowel have taken place. Temporizing is fatal. However, before an operation is resorted to we must be quite certain that we are not dealing with acute peritonitis, appendicitis, or intestinal obstruction from other causes—with which diseases intussusception is most apt to be confounded.

DIFFERENTIAL DIAGNOSIS.

CHARACTERISTIC SYMPTOMS	INTUSSUSCEPTION	ACUTE APPENDICITIS	ACUTE PERITONITIS	STRANGULATION
Onset	Sudden	Variable	Variable	Sudden
Tumefaction, its seat and nature.	Most frequently ileocecal region, occasionally round tumor in rectum	McBurney's point. Rigidity of abdominal wall	Distributed throughout abdomen, also local exudation	Local distention of bowel. Chiefly at abdominal rings
Tympanites	Moderate	Absent	Pronounced	Slight
Abdominal pain	Intense, general	Moderate, local	Marked, general	Severe, general
Constipation	Late, preceded by frequent mucohemorrhagic stools	Early	Late	Early
Fever	Slight	High	High	Slight
Collapse	Early	Late	Early	Early

When the services of a competent surgeon are not obtainable, an attempt may be made to reduce the invagination by copious injections of warm (100° F.) water into the bowels, or by air inflation.

Copious injections.

For the water injections an ordinary fountain syringe with a rectal tube, suspended about four feet above the level of the patient's pelvis, answers the purpose. Two to four quarts of water should be used. During this procedure the patient should be kept on his back with his buttocks raised about one foot above the level of the shoulders. Occasional inversion of the child is useful.

Morphine and atropine.

For the relief of pain and arrest of undue peristalsis, morphine and atropine hypodermatically; to check vomiting, lavage; to combat collapse, stimulants and external heat. Liquid, easily digestible food to sustain nutrition. Complications arising should be treated according to indications.

In view of the obscure causes of this affection, very little can be accomplished in the way of prophylaxis. Avoidance of habitual constipation, drastic purgatives, and of violent exercise (rapid up-and-down motion) may prove efficient prophylactic measures. The relationship between invagination and polypoid growths still lacks authoritative confirmation.

APPENDICITIS, TYPHLITIS, PERITYPHLITIS.

Frequently overlooked.

Until recently the prevalence of appendicitis in early childhood was not taken very seriously by the profession at large, and, hence, either because of this skepticism, or for want of understanding of the pathology of the disease, a great many cases of acute or chronic appendicitis were either overlooked, erroneously diagnosticated, or newly discovered as "food fever," "cyclical vomiting," and the like. Nowadays, the occurrence of appendicitis in children and even in sucklings is no longer doubted. On the contrary, in view of the frequency with which the vermiform process is found implicated in the course of severe infantile gastrointestinal disease, and its tendency by its relatively greater length and width to favor lodgment of foreign bodies (such as fecal concretions, worms, etc., which act as sources of infection), there is ample reason for the belief that appendicitis is—at least—as common in children as in adults. As in the latter the severity varies from simple inflammation to fatal gangrene, depending of course upon the type and virulence of the

Common in children.

causative bacteria and the promptness with which it is discovered and treated.

Inflammation.
Catarrha

Pathologically the simplest form of appendicitis consists of a catarrhal inflammation of the appendix. Its mucosa and follicles are reddened and swollen, and their secretion more abundant than normal. The lymphatics of the walls and of the surrounding structures are congested. Gradually the submucous and serous layers become involved and the appendicular lumen narrowed. In mild cases the obstruction in the appendix subsides, allowing the escape of the mucous and bacterial contents, and, with the exception of slight thickening and adhesions, rapid restitutio ad integrum takes place.

Ulceration.
Suppurati

In more severe cases the obstruction continues, the appendix becomes more and more distended, the mucous secretion purulent, the muscular coat—owing to its effort to expel the appendicular contents—thicker, hypertrophied, while the mucous membrane, as a result of pressure from within the appendix, undergoes gradual atrophy and ulceration. Even in this stage of the disease spontaneous recovery by encapsulation and absorption of the abscess is still possible.

Abscess formation.

In the majority of instances, however, instead of being absorbed, the purulent content of the appendix gradually, or rapidly, increases in quantity, and finally perforates the overdistended, more or less ulcerated appendix. The escaping pus finds its way where there is least resistance—into the cecum, small intestine, rectum, urinary bladder, gall-bladder, diaphragm or into the free peritoneal cavity. The pus may also penetrate into the retroperitoneal cavity or externally, usually in the right iliac region.

Gangrene.

Sometimes the inflammation is almost from the start so intense that perforation and gangrene of the appendix, and escape of its virulent contents into the peritoneal cavity occurs before a diagnosis can at all be arrived at. In these cases it is not rare to find also old inflammatory adhesions, indicating that the patient had once before gone through an attack of appendicitis (recurrent appendicitis), which probably was mild and has escaped attention.

The great variability in the course and termination of the aforementioned pathological processes can readily be explained primarily by the difference in the virulence of the causal bacteria, no single type of which having thus far proved to be the specific etiologic factor of appendicitis as a whole or of any of its forms. The bacteria found in the inflammatory products of the disease are principally streptococci, staphylococci, the bacterium coli, the pneumococcus, influenza bacillus, etc. It is not at all uncommon for appendicitis to develop in connection with pneumonia, influenza, gastroenterocolitis, etc., thus tending to prove its infectious character. Prominent etiologic factors also are: Retention of fecal concretions, foreign bodies (pins, fish-bones, cherry-stones, or orange-pits), intestinal worms, traumatism, exposure to cold and wet, etc. Male children (being more often exposed to the last named causes?) are more frequently attacked by appendicitis than female. Constipation and dyspepsia serve as predisposing causes.

Bacteria in appendix.

Predisposing causes.

Acute appendicitis may set in very suddenly or be preceded by premonitory signs consisting of frequently recurring attacks of dyspepsia, with colic and constipation. It is quite probable, however, that the dyspeptic symptoms are in reality the manifestations of recurrent catarrhal appendicitis of very mild type. Appendicitis once established, the little patient stops eating, is nauseated, vomits, and cries because of pain in the abdomen. The latter is more or less rigid. The anorexia is usually complete, and, if the child is forced to eat, the food is sooner or later ejected. Infants may continue taking the bottle or breast, to quench the ever-present thirst. In very mild cases nausea may replace the vomiting, but the latter symptom is always present in moderately severe cases and is quite severe in grave appendicular involvement, especially when the peritoneum is implicated. Pain, spontaneous and on pressure, is invariably present during an attack, but it varies greatly in severity irrespective of the pathologic condition of the appendix. Sudden cessation of pain is supposed to signify mortification of the underlying structures, and, hence, looked upon as a bad omen. Young children are usually unable exactly to localize the seat of the pain they are suffering from; little reliance, therefore, should be placed upon their localization. On the other hand, pressure pain can readily be elicited, and, as a rule, is most intense over the region of the appendix, which in children does not always correspond with "McBurney's point"—the appendix is often situated either higher up or lower down in the pelvis. Sometimes even infants indicate the presence of pressure pain by attempting unconsciously to ward off the examining hand, by placing their little

Vomiting.

Abdominal pain and rigidity.

Pain on extension of leg.

Situation of appendix.

hands over the most painful spot. Rigidity of the abdominal wall forms a pathognomonic sign of the disease and proves of great help in the diagnosis of appendicitis to one familiar with the peculiar sense of resistance of the abdominal wall to pressure. As a rule, the abdomen is distended, but it may also be contracted and as hard as a board. On gentle palpation the rigidity yields sufficiently to permit the detection of tumefaction —the underlying thickened appendix in catarrhal appendicitis, or the variously sized, hard or doughy, immovable mass, in appendicular abscess. In some cases the tumefaction may be seen to project beyond the normal level of the skin, and be felt in the rectum, a digital examination of which therefore should never be omitted. Appendicitis is ordinarily associated with complete constipation; the attack may, however, be ushered in by diarrhea, or, rather, *pseudo*-diarrhea—since the stool is derived chiefly from the lower part of the colon, superinduced by the sudden irritation within and about the appendix. As the disease advances, in consequence of pressure by the growing tumefaction in the pelvis, there may be severe tenesmus (as well as strangury) with or without a bloody discharge,—a symptom which is very apt to mask the diagnosis. The temperature is moderate, from 101° to 103° F. in catarrhal appendicitis, and as high as 105° F., in abscess formation. In favorable cases the pulse and respiration agree with the rise or fall of the fever. Low temperature with a high, feeble pulse is considered a bad omen, an indication of profound sepsis.

Tumefaction.

Pseudo-diarrhea and constipation.

Diagnosis.—Cases presenting the aforementioned typical symptoms of appendicitis can be diagnosed as readily in the child as in the adult. In fact, owing to the thinness of the infantile abdominal wall, and the proportionately large size of the appendix, it is usually not difficult to palpate an inflamed appendix unless it be—as it sometimes happens—misplaced somewhere beyond the reach of palpation. On the other hand, there is often considerable difficulty to differentiate an appendicitis pursuing a very violent course with marked tympanites, shock and collapse, from a grave attack of acute gastroenterocolitis, typhoid with perforation, intussusception, hernial strangulation and the like.[1] Even in such cases careful analysis of the typical symptoms of the respective diseases rarely fails to lead to a correct diagnosis.

Differential diagnosis.

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[1] See page 213.

Course and Termination.—The severity or mildness of the onset of an attack of appendicitis bears no positive relation to the further course of the disease. After the inflammatory process has, so to say, localized itself, which occurs usually within the first twenty-four or forty-eight hours, in the majority of instances the physician is able to conclude what sort of a case he is dealing with. By that time he will find that in *catarrhal* appendicitis the vomiting has partially or entirely ceased, the pain diminished, the abdominal rigidity lessened, and the tumefaction become less palpable. The child is able more easily to move about in bed, to have a few hours of comfortable sleep, occasionally to expel flatus, and to express a desire for food. Uneventful recovery may now take place within ten days, *i.e.,* as far as subjective signs are concerned. In the majority of cases some morbid anatomical changes remain in the appendix and adjacent structures, *e.g.,* inflammatory adhesions, kinking, constriction of the lumen, etc. The region of the appendix thus remains a *locus minoris resistentiæ* for life subject to recurrent attacks of inflammation and its sequelæ.

Localization of inflammation.

Sometimes after an apparently benign course of a few days' duration, either without discernible cause or as a result of gross error in diet, undue exercise, and the like, there is a sudden change for the worse. The symptoms spoken of to occur with the onset return, sometimes even in more pronounced form: the patient vomits, has chills, headache, severe pulling and throbbing pain in the abdomen. The temperature rises, the pulse increases in frequency and tension, respiration is quick but superficial (the patient is afraid to take a deep breath or to cough owing to the increase of the pain with the descent of the diaphragm); the child is restless and sleepless, lies principally on his back with his right leg flexed (attempt to extend it aggravates the pain) and cries with pain on being moved about. Palpation reveals a distinct oblong tumor, the distended appendix, which is very tender and gives rise to a gurgling sound on pressure. If the disease is not checked by operation the indurated mass enlarges, loses its circumscribed character, becomes more doughy in consistency, and dull on percussion, in short presents unmistakable signs of a fluid content—an abscess. This clinical picture of *suppurative* appendicitis does not by any means follow only the catarrhal variety; on the contrary, quite often it is in full development within the first two or three days of the disease, and, if the

Recrudescence of attack.

Abscess formation.

abscess is not promptly opened, it bursts, often giving rise to general peritonitis and quick death. More rarely the accumulation of pus occurs very slowly and gradually and even remains in abeyance for a period of weeks or months, during which time the abscess becomes walled off from the general peritoneal cavity by inflammatory adhesions, and may finally be absorbed, or with recurrent attacks of appendicitis perforate the sac and wander in any of the neighboring structures, sooner or later leading to the grave symptoms previously spoken of.

Encapsulation of abscess.

In another group of cases—*fulminating, gangrenous* appendicitis,—the symptoms are extremely alarming right from the beginning of the attack. In the midst of apparent good health, or preceded by slight malaise, vomiting, colic, prostration and collapse, follow one another in rapid succession, and often without palpable local appendicular tumefaction, or any other signs pathognomonic of appendicitis; the typical picture of general, septic peritonitis is in its full sway,—sometimes within twenty-four hours (usually after from three to five days) carrying the little victim to the grave. In such cases post-mortem examination reveals either pre-existing appendicular abscess, sudden rupture of the pus-sac, and diffuse infection of the peritoneum, or sloughing of a gangrenous appendix, involvement of adjoining blood-vessels (thrombophlebitis) and general sepsis (pyemia).

Fatal symptoms.

Post-mortem findings.

In view of the uncertainty of the course of the disease, every case of appendicitis should sooner or later be operated upon. This opinion is in accord with that held by the best clinicians of this age. The profession is still divided, however, on the question of *time*, when operative procedures prove most propitious for the patient's uneventful recovery. In solving so difficult a problem, the physician must be guided: 1, by the condition of the patient, and 2, the progress of the disease.

Time for operation.

1. *The Condition of the Patient.*—It certainly would be folly to operate on a child in a moribund condition, or on one synchronously suffering from a systemic fatal disease *per se, e.g.*, miliary tuberculosis, diabetes, grave heart or kidney disease, and the like. An operation should be deferred in infants under six months of age because of the lack of resistance of the patient, and in view of the fact that in very young infants spontaneous recovery (at least temporary!) by absorption of the pus, or rupture of the abscess in the rectum are by no means rare.

Contra-indications.

2. *Progress of the Attack.*—Mild catarrhal appendicitis, with

the first attack, progressing favorably during the first four days, may be left alone until the quiescent stage, when the appendix should be removed. Severe or recurrent catarrhal appendicitis failing to improve after the fourth or fifth day, or showing incipient symptoms of suppuration (increased leucocytosis!) should be operated upon at once, or if for some reasons an operation cannot be undertaken, treated medically for a week or ten days longer, until the abscess has become circumscribed and encapsulated, when an operation should be performed without further delay. The same rule applies also to all cases of slowly developing suppurative appendicitis, the physician being constantly on the guard, however, for sudden threatening symptoms of perforation,—in the latter event demanding prompt surgical interference. Finally, an immediate operation is imperative in all cases of perforative and gangrenous appendicitis,—procrastination being almost invariably fatal.

Indications for immediate operation.

In advocating operative procedures I presuppose that a competent surgeon is within reach to perform the operation. Otherwise the patient will fare better under medicinal treatment, which though only palliative is nevertheless potent to tide over the acute symptoms—often to hold the patient alive long enough until the services of a competent surgeon can be enlisted. The discredit cast in various quarters upon the surgical success in appendicitis to a great extent is due to the fact that the statistics compiled to show the bad results of operative treatment of appendicitis embrace the work of the competent and incapable surgeons alike, failing to specify that the work of the incompetent is chiefly responsible for the mortality. It is one thing for a surgeon skillfully to remove a purely inflamed appendix, and quite another to be able successfully to meet emergencies in badly complicated appendicular abscess or gangrene! The sooner, therefore, the attending physician will appreciate the fact that in the majority of the promptly treated cases the prognosis of appendicitis depends solely upon the skill of the surgeon, the quicker will the mortality from this affection dwindle down to insignificance!

Results greatly depend upon the skill of the surgeon.

When a patient is seen early, it is advisable to administer one dose of castor oil or calomel with bicarbonate of soda, and to wash out the stomach and intestine,—to clean the alimentary canal of its contents. This should be followed by the occasional administration, in the form of suppositories, of very small doses of codeine or opium, to arrest peristalsis and to keep the child

Arrest of peristalsis.

perfectly at rest and free from severe pain. No medication by mouth! During the acute stage of the disease, the constant application of ice is useful to relieve pain and arrest rapid progress of the inflammation. Thirst should be relieved by *small* quantities of water or tea, and as long as anorexia exists, no attempt at forced feeding should be tolerated. An occasional teaspoonful of milk, beef-juice, or broth, will prove sufficient to sustain life for days. Any indiscretion in the diet is hazardous. More liberal feeding may be practised after subsidence of the acute symptoms, after repeated escape of flatus, or partly formed stool. Even then extreme caution is commended, limiting the dietary to slowly increasing quantities of milk, broths, albumin water; in older children fresh soft-boiled egg, milk toast, small portions of fine cereals, etc. For marked flatulence atropine hypodermatically. Stimulation by means of strychnine and normal saline solution, both subcutaneously, should be resorted to in accordance with indications. As the patient improves, medication in the form of stomachics, intestinal antiseptics and laxatives may be administered by mouth, and the supply of nutritious food increased, so as to heighten the patient's vitality for an early operation. Children convalescing from an attack of non-operated appendicitis should not be taken to any resort where a competent surgeon is not within immediate reach. Danger always lurks behind a diseased appendix.

No medication by mouth.

Starvation diet.

Delayed operation.

PERITONITIS ACUTA.

Acute, non-tuberculous, peritonitis is of rare occurrence in children. It is occasionally encountered as a result of direct violence or in connection with infectious diseases (*e.g.*, typhoid fever, scarlatina, diphtheria), appendicitis, pneumonia, vulvovaginitis and other pus foci. In the newly born it not rarely forms a partial manifestation of sepsis (*q. v.*).

Rare in children.

Acute peritonitis usually sets in with very acute symptoms: Excessive pain and tenderness of the abdomen, rapidly developing tympanites, at first diarrhea, later constipation, scanty urination or complete anuria; sometimes distinctly localized exudation; high fever, especially in the first few days, and feeble, rapid, and very poor pulse; dry and brown tongue, anxious and pinched expression of the face, and, as the disease progresses, collapse. The course of the disease varies. Hyperacute peritonitis ends fatally usually in two or three days; moderately

Intense pain, tympanites.

Collapse.

severe cases may last a week, and then either terminate in death or in gradual recovery. To the latter class belong also the cases in which the pus breaks through the umbilicus, rectum or bladder.

Grave prognosis.

At all events the prognosis is very grave. It is almost always fatal in the newborn, and in the cases resulting from intestinal perforation. Traumatic peritonitis offers the most favorable outcome, and local peritonitis with encapsulated abscess often yields to prompt and suitable treatment.

The treatment, of course, depends entirely upon the underlying condition. It is justifiable to recommend an operation (laparotomy) in all cases of acute general peritonitis which fail to respond to medical treatment within forty-eight hours, and in those resulting from perforation of an abdominal viscus (*e.g.*, intestinal perforation in typhoid).

Morphine and atropine hypodermatically.

Rectal tube.

The medical treatment consists of perfect rest for the body and immobilization of the intestine. This may be secured by the hypodermic administration of morphine (gr. $\frac{1}{60}$, for a child two years old) and atropine ($\frac{1}{1000}$), the application of an ice-bag or light turpentine stupes to the abdomen and discontinuance of any nourishment until vomiting has completely ceased. Vomiting is best arrested by lavage or minute doses of iodin. After arrest of vomiting feeding may very cautiously be resumed. Breast-fed babies may again be put to the breast and bottle-fed should receive small quantities of milk, gruel, beef-juice, Tokay wine, champagne, and, if improvement continues, light mixed diet. For excessive tympanites the long rectal tube may be tried, allowing it to remain *in situ* for hours at a time. Cases running a protracted course often do well on daily local inunction of ung. hydrargyri ($\frac{1}{2}$ a dram), and the iodids internally. Localized abscesses should be incised and drained. In slow convalescence, a sojourn at the seashore will prove beneficial. (For Tuberculous Peritonitis, see page 366.)

INTESTINAL WORMS.

Worms gain entrance into the human system chiefly through the ova—consumed with food or water, or carried to the mouth by means of the fingers. We distinguish the following varieties of worms:—

Oxyuris Vermicularis (Seat-, Thread-, or Pin-worm).— Small, white, thread-like, freely movable worm, one-fourth to one-half inch in length. Its chief seat is the

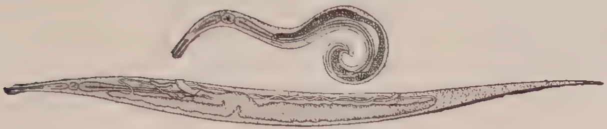

Fig. 55.—Oxyuris Vermicularis. Female and Male. (After *Leuckart.*)

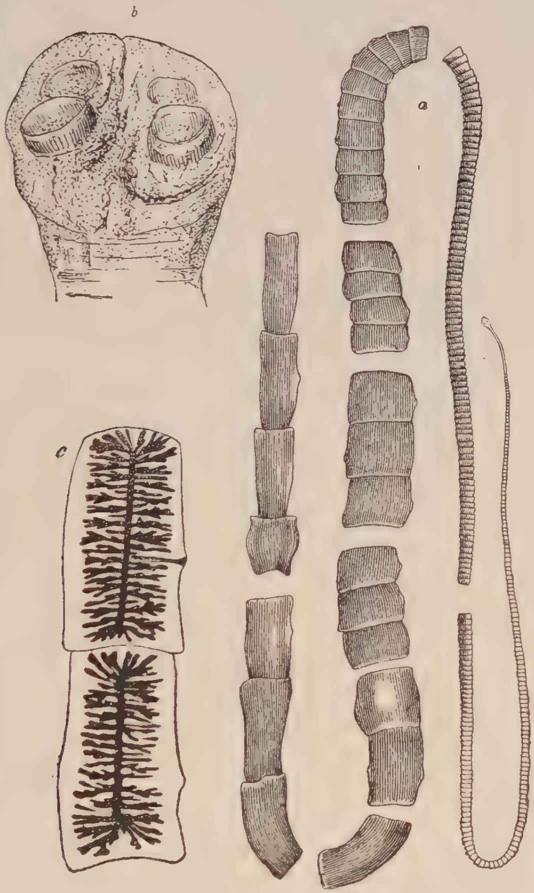

Fig. 56.—Tænia Saginata. *a,* Natural size of the worm at different sections. *b,* Head (with pigment canaliculi). *c,* Proglottides. (Partly after *Leuckart.*) (*Lenhartz.*)

Invades appendix.

rectum, where it causes intense itching. It may also infest the colon, cecum, appendix and vagina (vulvovaginitis).

Ascaris Lumbricoides (Roundworm).—Cylindrical, reddish gray in color, from four to ten inches in length. It resembles the earthworm in form. Its chief seat is the small intestine, but it may migrate to the stomach, gallbladder (icterus), throat, etc., in the latter event occasionally producing attacks of suffocation.

Throat.

Tæniæ (Tapeworms).—They are segmented worms of variable size. They inhabit the intestine and develop by budding.

(*a*) Tænia Mediocanellata s. Saginata, or the beef tapeworm. It is several yards long. The head presents at its middle a pit-like excavation and four anterior suckers.

(*b*) Tænia Solium, or pork tapeworm. It is shorter than the former. It is provided with four suckers, one proboscis, and a wreath of hooklets. After invading the human stomach the liberated embryos may wander to various portions of the body (skin, heart, brain, and eyes) and there develop into small vesicles (cysticercus) and lead to serious disturbances.

Skin, heart, brain, and eyes.

(*c*) Bothriocephalus Latus, or fish tapeworm. Several yards long, possesses about 3000 segments, a flattened head with two shallow suction grooves. May be the cause of severe anemia.

Severe anemia.

(*d*) Tænia Nana. About one inch long, possesses a head with four suckers and a wreath of hooklets. May cause stubborn diarrhea.

Stubborn diarrhea.

(*e*) Tænia Cucumerina s. Elliptica. From five to fifteen inches long; develops from swallowing dog-ticks that infest the hair of dogs and cats.

(*f*) Tænia Echinococcus. It inhabits the intestine of the dog. The latter transmits the ova to the human gastro-intestinal tract through the mouth by licking, etc. The embryos develop chiefly in the liver and lungs, forming cysts.

In liver and lungs.

Symptomatology.—In times bygone the laity looked upon intestinal worms as the source of all evil, and even the physician was frequently inclined to hold the same view. As a matter of fact, worms, with but few exceptions, rarely produce very serious

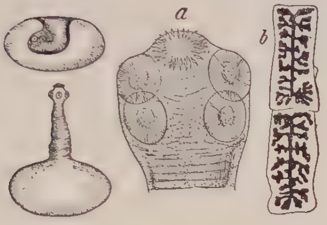

Fig. 57.—Tænia Solium. (After *Leuckart.*)

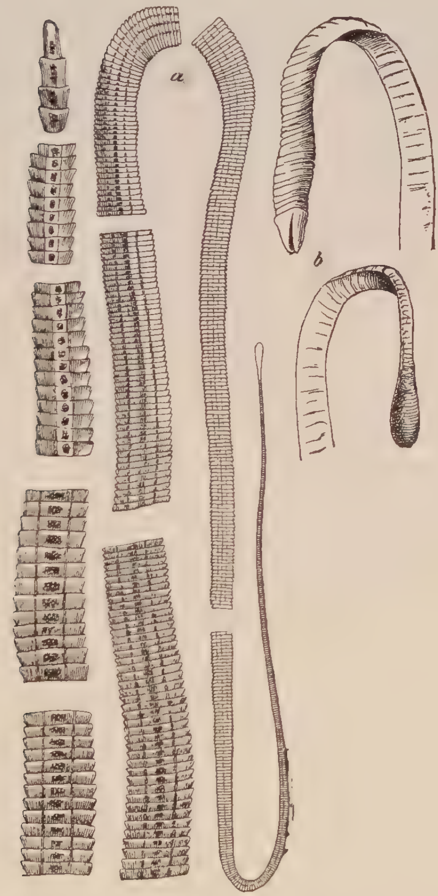

Fig. 58.—Bothriocephalus Latus. *a,* Worm, in sections; natural size. *b,* Head; lateral and front views. (After *Leuckart.*)

As a rule harmless.

disturbances. Indeed, numerous round- and tape-worms may infest the human intestines often without any indication of their presence until accidentally discovered in the stools. Among the signs which are otherwise said to indicate their presence are the following: A pale complexion, black rings under the eyes, *fœter ex ore,* capricious appetite, picking at the nose, recurrent urticaria, colic, headache, vertigo, apathy, mydriasis, pavor nocturnus, grinding of the teeth, and dry cough. Some authors claim to have observed divers neuroses, convulsions, chorea, trismus, epilepsy, amblyopia, strabismus, and the like. The majority of the reported cases of this sort, however, do not bear close scrutiny and are readily traceable to other causes. The actual harm done by some of the worms has been mentioned under each heading.

Pallor, capricious appetite.

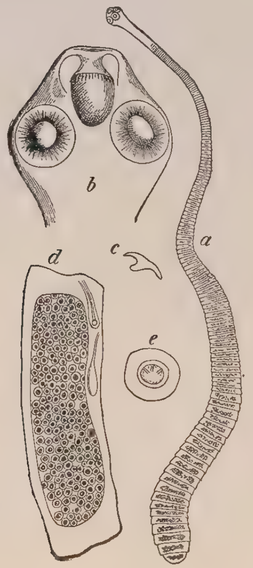

Fig. 59.—Tænia Nana. *a,* The whole worm (× 9). *b,* Head (× 50). *c,* Hooklet (× 300). *d,* Segment (× 50). *e,* Egg (× 125). (After *Leuckart.*)

Examination of stools and sputum.

Diagnosis.—The diagnosis can readily be made by macro- and micro-scopic examinations of the stools and sputum (echinococcus hooklets) for worms or their ova. The finding of intestinal parasites may be facilitated by the administration of anthelmintics.

Treatment.—Santonin and calomel act very efficiently in thread- and round-worms.

℞ Santonini,
Hydrargyri chloridi mite ....āā gr. vj | 0.4
M. et div. in pulv. no. vj.
Sig.: One powder to be given every morning, on an empty stomach, for a child 3 years old.

To expel tæniæ the following is a very useful combination:—

℞ Ext. fil. mar. æth. .... ʒiij | 12
Emulsi chloroformi .... ʒiv | 15
Emulsi amygdalarum ....q. s. ad ℥ij | 60
M. Sig.: Two teaspoonfuls as a dose for a child 3 years old, to be administered as follows:

The day before the diet should be restricted to fluids. In the evening the patient is given a few pieces of salt herring, followed an hour later by a purgative (castor-oil or calomel). The next morning the male fern should be administered on an empty stomach followed within half an hour by a dose of castor-oil or calomel. If only part of the tapeworm escapes, and the other part remains inside, the torn end should by means of adhesive plaster be fixed to the buttocks, and another dose of the anthelmintic administered until the rest of the worm has been expelled.

Mode of administration of male fern.

The effect of anthelmintics by mouth is greatly enhanced by enemas of soapsuds and turpentine (ʒss to Oj) or a decoction of quassia wood. Quassia is very useful in pinworms, especially if followed by local application of gray ointment. In older children the fluidextract of male fern may preferably be given in capsule form. The rare attacks of asphyxia from roundworms are best relieved by turpentine administered by mouth or by rectum.

Quassia in pinworms.

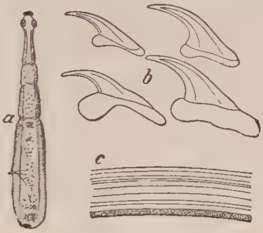

Fig. 60.—Tænia Echinococcus of the Dog. *a*, Tænia. *b*, Hooklets. *c*, Membrane fragment. (After *Leuckart*.)

ANKYLOSTOMIASIS. UNCINARIASIS
(Hookworm Disease).

Although prevailing in this country for many years past, this affection has only very recently, principally through the efforts of Dr. Charles W. Stiles, received due recognition as the "American murderer." It is practically endemic throughout the South, but is met sporadically also in other States of the Union.

Endemic in the South.

The disease is caused by the hookworm which infests the human body either through the mouth (by swallowing of infected water or food), and through the skin, especially the skin of the feet (the larvæ of the worm gradually entering the circulation), and ultimately settles in the upper portions of the small intestines.

The hookworm comprises two species: *Ankylostomum duodenale* (old-world species), which is endemic, especially in Italy and Egypt, and *Uncinaria Americana* or Necator Americanus (the

Ankylostomum.

Uncinaria.

new-world species). Both species measure from about ⅓ to ⅔ inch in length (the females somewhat larger than the males), but while ankylostomum carries on its head four hook-like teeth on the ventral side and two smaller vertical teeth on the dorsal side, the uncinaria has a dorsal pair of prominent semilunar plates or lips, and a ventral pair of smaller plates of similar nature.

By means of its armed mouth the worm fixes itself to the intestinal mucosa, producing minute erosions and hemorrhagic spots, and sooner or later a more or less severe catarrhal process in the alimentary tract. It is still a matter of diversity of opinion, whether the uncinaria feeds on the epithelial cells of the mucosa or upon blood. However this may be, the blood certainly undergoes marked changes, in severe cases, resembling the blood findings of primary pernicious anemia. Very soon other organs of the body are affected, especially the liver and spleen.)

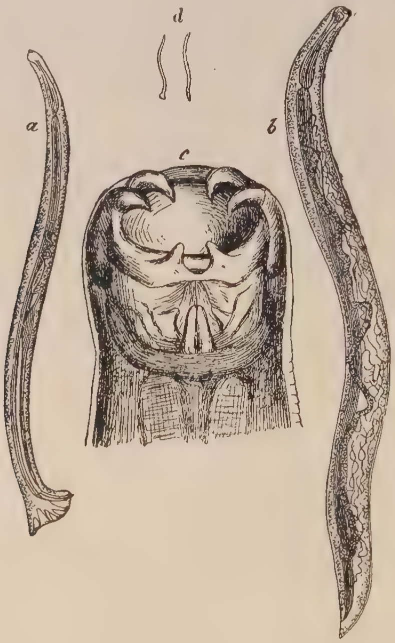

Fig. 61.—Ankylostomum Duodenale. *a*, Male. *b*, Female. *c*, Head. *d*, Natural size. (After *Leuckart.*)

Changes in the blood resembling pernicious anemia.

Post-mortem examination usually reveals fatty degeneration of the liver; softening of the spleen and paucity in lymphoid

Post-mortem findings.

elements; nephritic changes in the kidneys; pallor of the lungs; flabbiness of the heart, and anemia of the brain and effusion into the ventricles.

Stunted growth.

Pallor.

Pica.

Dropsy.

Hookworm disease is most destructive in the young. Children remain stunted in physical and mental development; they look old, tired, apathetic, and with puffiness of the face not rarely resemble cretins. The skin is sallow and the scleræ white or bluish-white. They suffer from palpitation of the heart, dyspnea, headache, dizziness, tinnitus, nausea, occasionally vomiting and abdominal pain. The appetite is either poor or voracious, often accompanied by a desire for unnatural food, leading to eating of earth, dirt, rags, etc. With increasing anemia there is frequently dropsy in the subcutaneous tissues and serous cavities—the edema often masking the emaciation and flabbiness of the body musculature.

Exhaustion.

Occasionally the disease runs quite a rapid course, within a few weeks ending fatally from exhaustion.

The diagnosis of hookworm disease is based upon a macroscopic and microscopic examination of the stools for the worm and its ova.

Thymol.

Thymol acts specifically in this affection. It may be administered in an emulsion with acacia or, in older children, in the form of capsules, the thymol crystals being first triturated with sugar of milk. The following mode of administration is recommended: Late in the afternoon the patient receives 2 grains of calomel (no castor-oil!), and the next morning 1 dram of Epsom salts. After the bowels have thoroughly acted 5 or 10 grains of the thymol are given on an empty stomach, and, if indicated, the dose repeated after an hour. The patient is kept in bed, without food, until late in the afternoon.

The feces should again be examined for uncinaria after the lapse of from two to four weeks.

Zu Osler -

CHAPTER VII.

Diseases of the Liver.

ICTERUS CATARRHALIS

(Catarrhal Jaundice).

Gastro-duodenal Catarrh.

CATARRHAL icterus (catarrh of the ductus choledochus) occurs as frequently in children over four years of age as in adults. It is comparatively rare in infants. As a rule, it is caused by and associated with gastroduodenal catarrh, and begins with coated tongue, anorexia, nausea, vomiting, and slight rise of temperature. Sometimes (*epidemic icterus*) the onset is sudden with high fever, apathy, delirium, headache and vomiting, so that before the appearance of the icterus cerebral disease is first thought of. In a day or two it is usually found that the urine is brownish yellow (bile-stained), the feces are gray and clayey, and the conjunctivæ, scleræ and skin yellow in color. This pathognomonic group of symptoms increases in intensity up to about a week, and then begins to diminish, first with clearing of the urine. The pulse is usually retarded, about seventy beats to the minute when the child is at rest. Palpation and percussion reveal tenderness over the stomach and liver, and occasionally some enlargement of the latter. This is particularly the case in catarrhal jaundice running a protracted course.

Regulation of diet.

No fats.

The prognosis is favorable and under suitable treatment the symptoms ordinarily subside within from ten to fourteen days. The treatment consists of restriction of diet to thin soups, albumin-water, skimmed milk, tea and toast, boiled fish or chicken, and similar, easily digestible food, free from fat (no cream, eggs or pastries!). Gradual return to a heavier diet. Medicinally, a few small doses of calomel and bicarbonate of soda, and daily intestinal irrigation (with 2 quarts of water, at 90° F.) will usually suffice to arrest the disease. Pancreatin, rhubarb and soda mixture, and sodium salicylate are useful remedies, and prolonged warm alkaline baths (one-half pound of bicarbonate of soda to the bath) hasten recovery in chronic cases.

℞ Acidi nitromuriat. dil.	ʒss	2
Elixir taraxaci (N. F.)	q. s. ad ℥ij	60

M. Sig.: ʒj in water, three times a day; in convalescent stage.

DISEASES OF THE PARENCHYMA OF THE LIVER.

Primary disease of the parenchyma of the liver is extremely rare in children under twelve years of age, since its principal cause—alcoholism—is practically unknown in young children. On the other hand, secondary involvement of the liver is not infrequently met in connection with syphilis, tuberculosis, chronic suppurative processes, malaria, rachitis, valvular heart disease, protracted gastrointestinal disease, and infectious fevers. In these conditions the symptomatology is the same as in adults.

In connection with chronic diseases.

CIRRHOSIS OF THE LIVER.

1. **Atrophic Cirrhosis.**—After a prodromic stage of several weeks, consisting chiefly of gastrointestinal disturbances, emaciation, tympanites, ascites, slight enlargement of the spleen, and dilatation of the abdominal veins gradually complete the clinical picture of the disease. The atrophy of the liver usually sets in insidiously, as a result of gradual hardening and contraction of the connective tissue. The course of the disease is shorter in children than in adults. Hemorrhages from the stomach and nose and into the skin not rarely occur toward the end of the disease, and progressive ascites hastens fatal termination.

Hemorrhages. Ascites.

2. **Hypertrophic Cirrhosis.**—This disease is characterized by considerable enlargement of the liver, pronounced icterus, very marked enlargement of the spleen, and protracted course. Ascites is absent until very late. The children usually remain stunted in growth. The liver is of very hard consistence.

Icterus.

3. **Congestive Cirrhosis (Cardiac Cirrhosis, Cardiotuberculous Cirrhosis).**—Pathologically it is characterized by hypertrophy of the liver and spleen, obliteration of the pericardium, and tuberculous pleuritis and peritonitis. Intense ascites forms the principal clinical symptom.

Pronounced ascites.

4. **Sugar-cake or Sugar-coated Liver (Pericarditic Pseudocirrhosis of the Liver—Pick's Disease).**—This form of liver disease is closely allied to the former variety. It is a progressive, incurable affection of unknown etiology.

No alcoholic stimulants.

Treatment.—As small quantities of spirituous liquors have proved to be the cause of quite a few cases of hypertrophic cirrhosis of the liver in children, it is essential to interdict its use in children, unless intended for temporary therapeutic purposes.

The iodids and mercury should be given a fair trial in all forms of cirrhosis irrespective of cause. The ascites may be relieved by tapping, if diuretics, cathartics and heart stimulants fail to do so. Bland diet. Sojourn at the seashore.

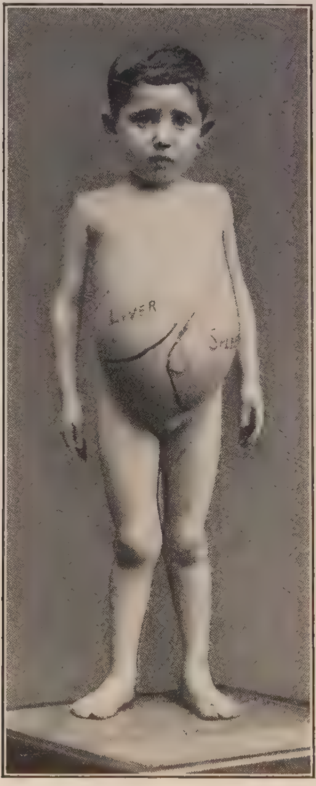

Fig. 62.—Amyloid Liver and Spleen at the age of 4 years. Followed multiple abscesses of skin. (*Sheffield.*)

ACUTE YELLOW ATROPHY.

Its course is very violent, sometimes ending fatally within a few days. The symptomatology is the same as in the adult: high fever, icterus, hematemesis, bloody stools, cerebral symptoms.

FATTY LIVER.

Anemia and emaciation are the principal symptoms. The liver is often moderately enlarged. The stools are grayish, pasty. The course is chronic.

AMYLOID LIVER.

It is often associated with amyloid degeneration of the spleen and kidneys, and secondary to some wasting disease, especially chronic suppurative processes in the bones and joints. The hepatic and splenic dullness is enlarged, but pain on pressure, jaundice, or ascites are absent, unless the portal circulation is interfered with by enlargement of the glands in the portal fissure.

Absence of ascites.

Attention to the cause and dietetic and hygienic measures may prove effective to arrest the degenerative process.

ABSCESS OF THE LIVER.

This condition is occasionally observed in children, most frequently as a result of extension of septic processes from neighboring structures, *e.g.*, suppurative appendicitis, phlebitis umbilicalis, typhoid or dysenteric intestinal ulceration. It may follow traumatism, invasion by roundworms, suppuration of echinococcus cysts, or of the mesenteric glands. The abscess may perforate into the thorax, intestines, or externally.

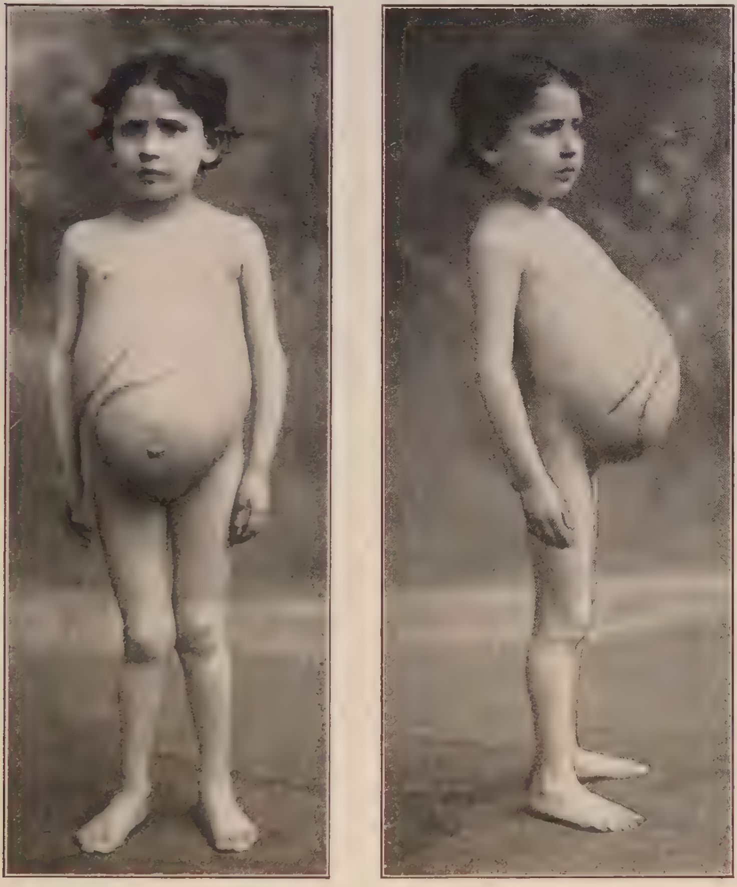

Fig. 63 Fig. 64

Fig. 63.—Amyloid Liver and Spleen at the age of 8 years. Same as Fig. 62. Fig. 64.—Side View. Patient otherwise well and happy. (*Sheffield.*)

Complication of suppurative processes.

Septic symptoms.

Symptomatology.—Chills, hectic fever, tenderness over the liver, marked gastrointestinal disturbance, slight icterus, enlargement of the liver, sometimes fluctuation and pus on aspiration.

Treatment.—Free incision and evacuation of the pus as soon as the diagnosis has been established.

Differential Diagnosis.

	Liver Abscess	Hydatid Cyst of the Liver	Pleurisy with Effusion	Solid Tumor of the Liver
Chills	Marked	Absent	Slight	Absent
Fever	Hectic	Absent	Moderate	Absent
Tenderness	Marked	Absent	Absent	Moderate
Icterus	Slight, early	Late	Absent	Marked, late
Fluctuation	Moderate	Pronounced "hydatid vibration"	Absent, diffuse flat area, uninfluenced by inspiration	Absent
Dullness	Highest in mid-axillary line	Highest in mid-axillary line	Lowest in mid-axillary line	Irregular
Aspiration reveals	Pus	Non-albuminous fluid with "hooklets"	Albuminous fluid which coagulates on boiling. Pus in pyothorax	Blood
Lung symptoms	Absent	Absent	Present	Absent

TUMORS OF THE LIVER.

Benign as well as malignant tumors of the liver are occasionally observed in young children and even in the newly born. Cystic degeneration is most common, and cases of carcinoma and adenocarcinoma, and more rarely sarcoma are on record. These growths should not be confounded with gumma of the liver—the effect of specific treatment being most decisive in the diagnosis.

Differentiation from syphilis.

CHAPTER VIII.

Diseases of the Respiratory System.

GENERAL REMARKS.

Inherent and acquired disposition to nasopharyngeal catarrh.

THE inherent frailty of the infantile respiratory tract is very conducive toward its morbidity. The nasopharyngeal passages being very narrow and winding—intended to halt air impurities and to moisten and warm the inspired air before its entrance into the larynx—functionate to their own detriment in localities where the air is dust-, smoke- and dirt-laden, and where atmospheric changes are many and marked. Thus, the child being unable to clear its nose, the detained foreign bodies irritate the delicate, highly vascular mucous membrane, before long forming a nidus for bacterial invasion. As we will see later "a cold in the head" is quite common in infants, and, while *per se* harmless in its immediate effect, is often serious in its remote results. The local congestion by its repeated recurrence produces a *locus minoris resistentiæ* not alone of the mucous membrane of the nose, but, by extension and persistence of the inflammatory changes (hypertrophy), of the pharynx and adenoid tissue as well. With ensuing nasopharyngeal obstruction breathing now proceeds principally through the mouth; the air no longer undergoes the preparatory process of filtration, moistening and warming, but reaches the larynx in its impure, irritating state, sooner or later giving rise to a catarrhal inflammation of the larynx and neighboring structures. This condition is soon aggravated by the continuous affluxion of foul nasopharyngeal secretion, and by the inability of the little patient to clear its throat by forceful expectoration. Furthermore, the thorax being short and narrow, its musculature thin and feeble, and the heart and thymus gland comparatively large, the more or less compressed lung is greatly hampered in free aëration and in ridding its distantly located portions of the obnoxious inflammatory products. Hence the pertinacity of apparently insignificant pulmonary lesions, the frequency of unresolved pneumonia and pyothorax, and the insidious development of asthma, atelectasis

Extension of inflammation to bronchi and pulmonary alveoli.

and emphysema. As the child grows older, the nasopharyngeal tract larger, the thoracic cavity more spacious and, synchronously, the respiratory function more forceful, there is a corresponding reduction in frequency and persistency of respiratory disease, notwithstanding, or, perhaps, because of the increased exposure of the child to atmospheric changes and infection.

DISEASES OF THE NOSE, THROAT AND EAR.

RHINITIS ACUTA

(Coryza).

Acute coryza is a frequent affection of childhood. It may occur primarily as a result of bacterial infection following exposure to thermic, mechanic or chemic irritation, or secondarily in association with measles, influenza, scarlatina and diphtheria.

Primary and secondary.

Primary coryza (with sneezing, slight rise of temperature, anorexia, etc.), while quite harmless in older children, is often very serious in infants. Here it usually begins with vomiting, fever, and sometimes convulsions and occlusion of the upper air passages by a mucous or mucopurulent secretion. Owing to thickening of the nasal mucous membrane there is partial or total obstruction to nasal breathing, giving rise to interference with suckling, dyspnea, and even acute attacks of asphyxia. The latter are prone to occur especially in the newly born who are not accustomed to breathe through the mouth and "swallow" the tongue.

Sneezing.

Hypersecretion.

Nasal obstruction.

Every case of acute rhinitis associated with severe local (membranous deposit) and systemic (vomiting, rapid loss of strength) symptoms should arouse the suspicion of being diphtheritic or scarlatinal in character.

Examination for diphtheria.

Acute rhinitis is not rarely complicated by otitis, laryngitis and bronchitis. The prognosis is generally good, although in young infants convalescence is slow.

Treatment.—Avoidance of exposure to all atmospheric changes. Cleansing of the nostrils by repeated instillation of a few drops of a 2 per cent. solution of bicarbonate of soda, alternated with lukewarm mentholated olive oil or albolene. Careful feeding, if necessary, by the spoon. As measures of temporary relief, we may recommend local applications of atropine (¼ per cent.), cocaine (1 per cent.), or suprarenal solutions ($^1/_{10}$ per cent.) and camphor and the salicylates and quinine internally.

More or less strict isolation of the patient. Attention to constitutional symptoms. Serum therapy whenever it is indicated (diphtheria.)

℞ Natrii salicyl.	gr. xij	0.8
Pulv. camphoræ	gr. iij	0.2
Chocolate	q. s.	

M. ft. pulv. no. vj.
Sig.: One powder every two hours for a child 3 years old.

RHINITIS CHRONICA

(Nasal Catarrh, Ozena).

Hypertrophic.

Atrophic.

It is characterized by marked congestion and thickening of the nasal mucous membrane and hypersecretion—*hypertrophic rhinitis*, or by atrophy of the various layers of the mucous membrane and foul-smelling incrustation—*atrophic rhinitis, ozena.* The latter form is rarely observed in children under ten years of age.

Syphilitic.

Chronic rhinitis is usually the result of repeated attacks of acute coryza or other affections of the nasopharynx associated with nasal hypersecretion and obstruction to free nasal breathing (adenoids). In the presence of foreign bodies in the nose it is usually unilateral. In the nursling it is often due to hereditary syphilis (*syphilitic rhinitis*).

Cleanliness.

Cauterization.

Treatment.—As all forms of chronic rhinitis by respiratory interference and secondary glandular infection give rise to more or less impairment of the constitution, the treatment of this condition should embrace local as well as general therapeutic measures. The nose and nasopharynx should be kept clean by antiseptic and oily sprays and the congestion allayed by painting the mucous membrane twice a week with silver nitrate (1 per cent.), tannin-glycerin (5 per cent. to 10 per cent.), etc. Excessive hypertrophy should be reduced by trichloracetic acid and similar caustics and, if these fail, by means of the galvanocautery or nasal scissors.

℞ Thymolis	gr. ij	0.15
Olei eucalypti	gtt. v	
Albolene	q. s. ad ℥ij	60.

M. Sig.: Nose-spray, to be used morning and evening.

EPISTAXIS

(Hemorrhage from the Nose, Nosebleed).

Bleeding from the nose may be due, primarily, to traumatism, external irritation of the mucous membrane from various

Numerous causes.

causes, foreign bodies, etc.; or may occur as a result of vascular excitement during the course of febrile, circulatory (especially after exertion) and pulmonary diseases; hemorrhagic affections, etc.

Treatment.—The treatment of epistaxis varies, of course, with the cause. In slight hemorrhage simple compression of the alæ nasi against the septum acts efficiently.

Astringents.

Sedatives.

In cases of moderate bleeding, sitting posture, head erect, with hands folded over the head, and ice application to the nose and nape of neck, or instillation of cold water (with some vinegar, alum or potassium permanganate) into the nose will usually suffice. If this fails, the nares should be packed as far back as possible with pledgets of cotton or gauze, dipped in a strong solution of alum, in peroxid of hydrogen, or suprarenal gland solution. In secondary epistaxis due to vascular congestion a small dose of morphine hypodermically in conjunction with the aforementioned measures will often act very promptly. As the last resort we turn to the post-nasal tampon which, as a rule, checks the hemorrhage unless hemophilia is the underlying condition of the bleeding, when the treatment must be directed chiefly against this affection (*q.v.*).

Cauterization of bleeding spot.

Detection of the local cause is very essential. Every visible bleeding spot should be cauterized with chromic or nitric acid or with the galvanocautery. Constitutional symptoms, if present, should receive prompt attention.

TUMORS AND FOREIGN BODIES IN THE NOSE.

Polypi.

Foreign bodies.

Mouth-breathing, snoring, and nasal speech are not due solely to adenoid vegetations or large tonsils. Not infrequently obstruction to breathing is the result of the presence of mucous polypi (soft, jelly-like), fibrosarcomas (hard and pedunculated) or foreign bodies. The latter are usually beans, pebbles, cherry-stones, and so-called rhinoliths. Sooner or later they give rise to (*unilateral*) foul, bloody discharges and implicate the lacrimal duct and Eustachian canal, and form a reflex cause of persistent, irritable cough, and asthmatic conditions. The diagnosis can readily be made by inspection.

Treatment.—Tumors should be removed with the cold snare, galvanocautery, or by torsion with a slender forceps. Bleeding may be arrested in the manner outlined under Epistaxis.

Foreign bodies if anteriorly situated can readily be removed by air inflation through the free side, or by means of a pointed forceps. If impacted farther back, it is preferable to dislodge the foreign body with a slender hook or forceps under cocaine and either extract it anteriorly or force it posteriorly into the nasopharynx.

Air inflation.

PHARYNGITIS ACUTA.

Acute pharyngitis is rarely primary (streptococcic infection) but quite frequently secondary in nature as a complication of acute rhinitis, tonsillitis, acute exanthematous affections, etc. Primary pharyngitis is ordinarily of short duration and manifested by dryness in the pharynx, pain in swallowing, and moderate rise of temperature. The pharynx is reddened, somewhat swollen, and often granular.

Primary and secondary.

Secondary pharyngitis will be considered in connection with the diseases it complicates.

Treatment.—Attention to the bowels, rest in bed, Priessnitz compresses to the neck and antiseptic sprays to the throat. Liquid non-irritating diet.

Symptomatic treatment.

PHARYNGITIS CHRONICA.

It may develop after repeated attacks of acute pharyngitis or as a result of extension of an inflammation from the adjacent structures. The posterior pharyngeal wall not rarely presents a deeply congested granular appearance, and here and there covered by a tenacious mucous deposit.

The affection is associated with more or less dryness in the throat, hawking and coughing. On examination the fauces appear swollen and relaxed, the tonsils hypertrophied, and the esophageal opening covered by a thick, grayish-white deposit.

Hypertrophy of fauces and tonsils.

Treatment.—Avoidance and removal of causes. Locally the parts must be kept clean by mild antiseptic sprays (Dobell's solution), and the swelling reduced by nitrate of silver (2 per cent.), or tannin-glycerin (5 per cent.) solutions. Change of air, iodid of iron, cod-liver oil, etc., are very helpful to effect a cure.

Nitrate of silver.

℞ Suprarenal solution (1:2000),
Dobell's solution ....āā ℥j | 30
M. Sig.: Throat-spray in acute or chronic pharyngitis.

ANGINA

(Sore Throat).

Tonsillitis Acuta, Amygdalitis, Quinsy.

Bacterial origin.

Chills, fever, pain and swelling.

Children under two years of age seem to present a decided immunity against tonsillitis. On the other hand, all forms of angina are extremely common in children over two years old. Those with a "catarrhal habit" are especially prone to contract the disease. Streptococci, staphylococci and pneumococci, among other micro-organisms, form the most frequent primary cause, and are productive of the usual symptom-complex which is characteristic of similar contagious and infectious diseases of childhood. Thus, the attack is ushered in suddenly with a chill, rise of temperature (with evening exacerbations), vomiting (in younger children) and sometimes convulsions. The younger the child the less conspicuous the dysphagia. Hence the importance of a routine examination of the throat in all febrile affections.

To avoid unnecessary repetition it is advantageous to classify tonsillitis in accordance with the tonsillar deposit as follows:—

Mucous exudation.

Angina Catarrhalis.—Redness and swelling of one or both faucial tonsils and adjacent tissues. Thin mucous exudation.

White dots.

Angina Follicularis.—The deposit begins as one or more white, small pellicles upon the middle or anterior portion of the tonsil. The white dots, at first distinctly isolated, soon coalesce to form yellowish- or greenish-white elevated patches. These are removable without profuse bleeding, and reform slowly.

Abscess "point."

Angina Parenchymatosa (Quinsy, Peritonsillar Abscess).—The tonsil (usually one) and peritonsillar tissue are greatly enlarged, often displacing the uvula. It is bluish in color and doughy in consistency. The deposit, at first white, gradually turns yellowish-green, resembling the "point" of an abscess. Pus on puncture.

Vesicles.

Angina Herpetiformis.—The deposit begins with minute vesicles, which tend to burst and leave behind superficial ulcers. This form of amygdalitis usually involves both tonsils and is at times complicated by stomatitis.

Angina Gangrænosa (Necrotica).—The tonsils are moderately enlarged and almost completely covered by a greenish-yellow, continuous, deposit surrounded by a red zone. The exudation if removed leaves behind a deeply seated ulcer. The deposit often spreads from one tonsil to the other by way of the anterior pillars, palatine arch and uvula.

Large deposit.

Angina Ulcerosa (Vincentii).—It greatly resembles the latter but is usually limited to one tonsil, and occasionally presents a pseudomembrane. Vincent's bacillus in pure culture is almost always found in the exudation.

Vincent's bacillus.

The course of the different varieties of tonsillitis varies but slightly. After subsidence of the acute initial symptoms previously spoken of, the disease assumes a much milder aspect, except as to prostration, pain in swallowing and evening exacerbations of the fever. The latter ranges between 102° and 105° F. and is especially high in follicular tonsillitis. More or less marked lymphadenitis is present in all forms of angina, and in accord with the tonsillar involvement it is either unilateral or bilateral. Parenchymatous angina is not infrequently associated with *pseudo*-torticollis, and pain on moving the jaws is present also in the other forms of the affection.

Torticollis.

In uncomplicated cases recovery is the rule in from three to ten days but quite a number of deviations from the usual course are observed. Tonsillitis is not rarely the forerunner of true diphtheria or rheumatic affections with their respective complications, and cases are on record where it proved to be the source of general septic or pyemic infection.

Rheumatism.

Differential Diagnosis.—Angina may be confounded with influenza, glandular fever, diphtheria and scarlatina. In *influenza* the exudation is slight and not strictly limited to the tonsils; adenitis is comparatively rare. Furthermore, influenza is characterized by the simultaneous presence of respiratory, digestive, and often nervous phenomena, while in tonsillitis throat symptoms predominate. *Glandular fever* differs from tonsillitis by the comparative absence of tonsillar manifestations and preponderance of glandular swelling (also of the bronchial, esophageal and retroperitoneal glands). The distinction between severe cases of tonsillitis and moderately severe forms of diphtheria without a bacteriological examination is often very difficult in the first twenty-four hours of the disease. In pharyngeal *diphtheria* the pseudomembrane appears as a small uneven, grayish white, slightly elevated patch upon the inner tonsillar or

Differentiation from influenza, glandular fever and diphtheria.

faucial surfaces of the throat. The deposit augments by rapid spreading, within a few hours reaching the posterior wall of the pharynx and adjacent structures. The surrounding, uncovered areas are grayish in color, due to overcrowding of leucocyte-nuclei and mucus beneath. The tonsils are moderately large in size, but the submaxillary glands are large and hard, assuming the shape of a large walnut and bulge conspicuously forward. The deposit, if removed, leaves a raw, bleeding surface and rapidly reaccumulates. This clinical picture differs materially from that of tonsillitis and often proves useful in arriving at a correct diagnosis. Tonsillitis with and even without erythema may be mistaken for *scarlatina* and a differential diagnosis is sometimes impossible until a few days after beginning of the attack.

Differentiation from scarlatina.

Antiseptic gargle.

Treatment.—In view of the possible serious complications tonsillitis should be arrested at its inception. The following mixture should be used every two hours as a local application—undiluted, by means of a cotton swab in young children, or diluted 1 to 20 of water, as a gargle, in older ones:—

℞ Acidi carbolici	ʒss	2.
Pulveris camphoræ	gr. x	0.6
Alcoholis	ʒij	8.
Glycerini q. s. ad	℥ij	60.

M. Sig.: One teaspoonful in twenty of water as a gargle every two hours, etc.

Salicylates.

Evacuation of pus.

For the relief of pain cold Priessnitz's compresses or an ice collar to the neck and salicylates internally. The latter is intended also to guard against rheumatic affections. In angina parenchymatosa if suppuration is inevitable it should be hastened by hot applications and the abscess opened early. Irrigation of the throat. Rest in bed, liquid diet, plenty of water. Avoidance of transmission of the disease. (See also "Diphtheria," page 296.)

HYPERTROPHY OF THE TONSILS.

Secondary.

Chronic enlargement of the tonsils often develops after repeated attacks of angina or pharyngitis, not rarely follows scarlatina or diphtheria and is frequently associated with adenoids. When the tonsils become so large as to obstruct respiration, the same symptom-complex makes its gradual appearance as is pathognomonic of adenoids. As in the latter anomaly, removal of the hypertrophied tissue is the only actual cure, and unless con-

Danger of hemorrhage.

traindicated by hemorrhagic diathesis, should be undertaken with the aid of a tonsillotome—the earlier the better, since the presence of more or less degenerated tumors acts not only as a cause of a number of reflex phenomena (*e.g.,* enuresis), but as a harboring place for divers pathogenic bacteria, including the tubercle bacillus.

Tonsillotomy is usually performed in the following manner:—

Tonsillotomy.

The patient is placed on a table (if an anesthetic is to be used) or seated on the lap of an assistant or nurse. The arms are immovably fixed by means of a wide towel or sheet. The tonsillotome is introduced into the mouth like a tongue depressor and turned sideways and pressed against the base of the hypertrophied tonsil so that its summit protrudes through the circular opening of the tonsillotome. With the tonsillotome thus fixed and the thumb of the operator in the handle of the blade the latter is firmly driven through the gland.

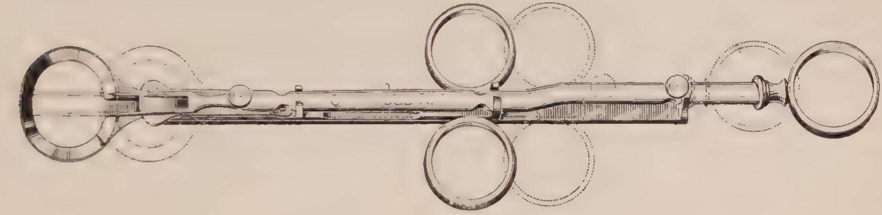

Fig. 65.—Tonsillotome.

The same procedures are repeated for the other tonsil.

Arrest of hemorrhage.

Slight bleeding calls for no treatment. Profuse hemorrhage should be promptly checked by local use of ice-water, peroxid of hydrogen, adrenalin (1:1000), local pressure, or other therapeutic measures generally employed in local hemorrhage.

ADENOID VEGETATIONS

(Hypertrophy of the Nasopharyngeal or Luschka's Tonsil).

The mucous membrane of the rhinopharynx is normally rich in lymphoid or adenoid tissue which bears the name of nasopharyngeal or Luschka's tonsil. Like the faucial tonsils the latter is subject to frequent attacks of inflammation with secondary hypertrophy. Whenever the hypertrophied adenoid tissue assumes such proportions as to more or less fill the nasopharyngeal space and obstruct nasal breathing, a pathognomonic clinical syndrome develops which, though apparently insignificant in its lesion, is often very serious in its immediate and remote consequences.

The clinical picture unfolds gradually, almost insidiously, growing more pronounced from time to time as the patient "catches cold." The child is unable to clear the nasopharynx, and the retained irritating nasal discharge helps to swell the adenoid tissue and to obstruct the rhinopharynx. The child is thus forced to breathe through the mouth. As immediate results we find that it keeps the mouth open, sleeps restlessly with the mouth open and as a rule snores heavily. He is frequently awakened by extreme dryness of the throat, and a croupy, harassing cough. In the morning he is tired, complains of headache, is drowsy and apathetic. His speech is dull, nasal (m and n sound like b and d) and hesitating, and sometimes stuttering.[1]

Open mouth.

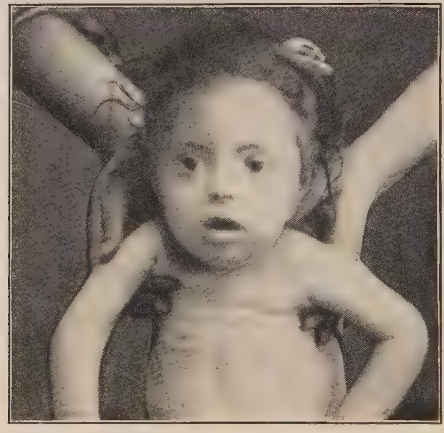

Fig. 66.—Adenoids. Note typical idiotic face. (*Sheffield.*)

Snoring.

Apathy.

Stuttering.

Were it possible to bring these little sufferers under proper treatment at this stage of the disease, quick and uneventful recovery would be the rule. Unfortunately, however, the laity, nay, the physicians as well, rarely think these symptoms of sufficient gravity to necessitate medical and particularly surgical intervention. The deplorable condition is therefore allowed to proceed and the tumor to spread and sprout. The sequelæ appear in rapid succession. The labored breathing sooner or later produces deformity of the thorax (pigeon breast) and often curvature of the spine. Owing to non-participation of the nose in respiration there is gradual atrophy of the levators alæ nasi et labii superiores, the depressors alæ nasi, and the septum mobile. The nose becomes pinched and pointed, the external angle of the eye deeper than the internal, the lower lip droops, the lower jaw sinks down, and the face assumes that dull, fixed and irresolute expression which is so characteristic of adenoids. In addition to this, hearing is impaired as a result of secondary catarrhal inflammation of the Eustachian tube, etc. The child is absent-minded and dull of perception, does poorly at school, and becomes the target for abuse and corporal punishment by

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[1] It should be remembered, however, that the presence of adenoids does not necessarily produce the typical symptoms of the disease. It all depends upon the proportionate size of the tumor to that of the rhinopharynx.

Labored breathing.

Deformity of thorax.

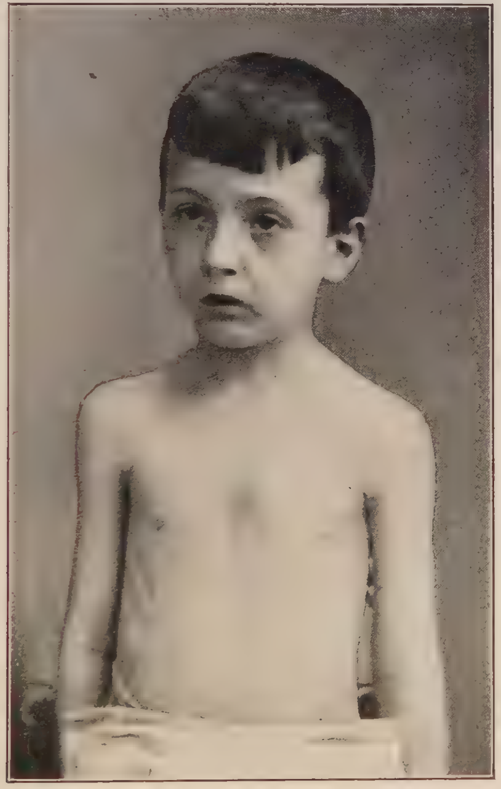

Fig. 67.—Adenoids. Note funnel-shaped chest. (*Sheffield.*)

Idiotic appearance.

Mental backwardness.

teachers and parents—all for no fault of his. When brought to the physician—often chiefly on account of impaired hearing—the diagnosis can readily be made by mere inspection. Such a superficial examination, however, should not be relied on, as similar symptoms are produced by nasal obstruction from other causes (deformities, growths, foreign bodies, etc.). Inspection of the mouth reveals the bony palate high and narrow, leaving insufficient space for the teeth and causing their displacement. The faucial tonsils are greatly enlarged (in about 25 per cent. of the cases), the posterior pharyngeal wall is granular, and, with the velum palati raised, often shows the distal ends of the adenoid vegetations. Rhinoscopy confirms the presence in the nasopharyngeal space of a pale-red, smooth, soft tumor which sometimes resembles a mass of earthworms. It bleeds readily. The

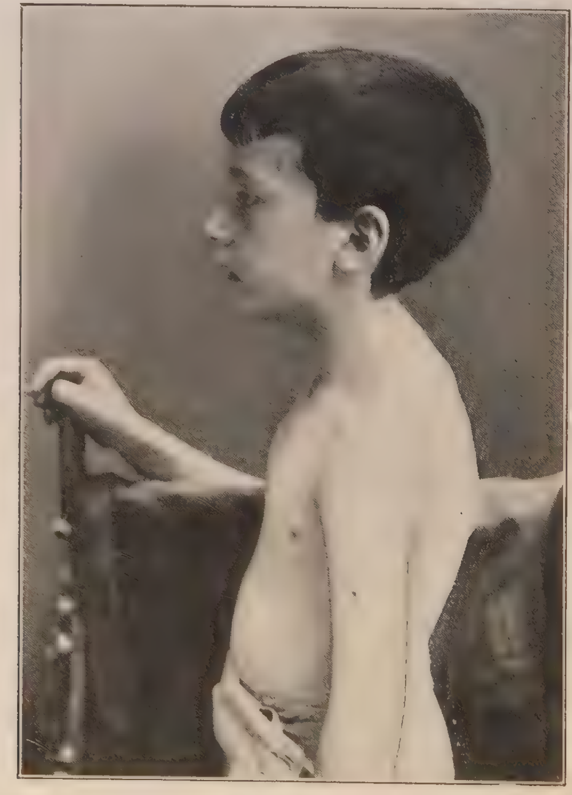

Fig. 68.—Adenoids. Note spinal curvature. (*Sheffield.*)

Deformity of palate.

Mass in rhinopharynx.

diagnosis is further corroborated by palpating with the finger the soft masses blocking the rhinopharynx, or by nipping off a small portion of the adenoid vegetations by means of an adenoid forceps introduced behind the velum palati.

The diagnosis once established the treatment should be prompt and energetic. *Mild cases in their early stages* may be arrested at their inception by scrupulous cleanliness of the nasopharynx, local applications of Lugol's solution or 2 per cent. of nitrate of silver, change of air, outdoor exercise, cold shower baths, and hematinics and alteratives internally. These procedures should also be followed in cases with *hemorrhagic diathesis* where an operation is contraindicated for fear of uncontrollable bleeding, and in those associated with other grave affections. In all other cases removal of the adenoids is the only actual cure, and should be undertaken as early as possible. The mode of procedure varies with each individual case. In young children under three years of age the operation may be performed without an anesthetic, in sitting posture; in older ones or in those who are hypersensitive to pain and shock preferably under primary anesthesia with ether (drop by drop method), ethyl ether or nitrous oxid gas, in recumbent posture. The child's arms are fastened to the sides of the thorax by a wide towel, and his jaws are separated by a mouth-gag placed between the left upper and lower teeth. The operator stands on the right side of the patient and introduces the adenoid curette sideways into the latter's mouth and passes it beneath the soft palate and up along the anterior wall until he reaches the vault of the rhinopharynx. The physician then implants the cutting edge of the instrument into the adenoid mass and makes a firm semicircular movement, directed backward, downward and forward. One such movement usually suffices to remove the tumor. It may be followed up, however, by a few lighter, similar strokes, to smoothen the rough edges. The patient is then turned on the side to allow the blood to drain into a basin. This may be facilitated by the injection of ice-cold water through the nostrils. After arresting the more or less profuse hemor-

Cleanliness.

Danger of hemorrhage.

Careful anesthesia.

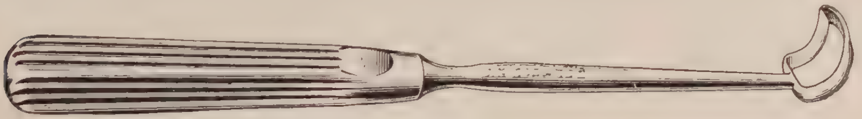

Fig. 69.—Adenoid Curette.

rhage, which always accompanies the operation, the child is put to bed for a few hours until he has regained full consciousness and kept indoors for a day or two on a non-irritating, cool, liquid diet.

After-treatment.—To prevent the recurrence of the adenoids, which is prone to take place in children with a tendency toward glandular hyperplasia, it is advantageous to instill into each nostril a few drops of Lugol's solution, once every other day for a period of about four weeks, and to use an oily antiseptic spray for several weeks thereafter. This procedure will prevent also adhesions between the cut surfaces and the soft palate. Delicate children should be put on syrup of the iodid of iron, cod-liver oil, etc. To regulate nasal breathing it is often necessary by means of a bandage to keep the mouth closed, especially at night, and to take prolonged breathing exercises with closed mouth. Impaired speech sometimes calls for instruction in speaking or, in the event of a paretic condition of the velum palati arising from inactivity, for treatment by electricity or tonics. In the majority of instances, however, the operation is followed by immediate restitutio ad integrum. All reflex symptoms and to a great extent even the deformities of the thorax subside rapidly.

Local treatment.

Bandage over mouth.

Tonics.

DANGERS AND ACCIDENTS ATTENDING ADENOID OPERATION.

Simple and harmless as the operation is under ordinary conditions, it is not always free from danger. As in more serious operations the possibility of fatality from the effect of the anesthetic or infection is gravely to be borne in mind and the frequency of primary or secondary—occasionally fatal—hemorrhage should engage the constant attention of the operator.

Anesthetic.

Sepsis.

Hemorrhage.

To obviate untoward complications all such preparations should be made as are customary with capital operative work. Ethyl chlorid and ether (drop by drop method) should be the anesthetic of choice, and primary in preference to deep anesthesia. The instruments to be used should be carefully sterilized, and the field of operation and everything coming in contact with it rendered as aseptic as possible. Before beginning the operation the surgeon should test the efficiency and entirety of his instruments, and see to it that he is amply supplied with all such drugs (peroxid of hydrogen, suprarenal gland in solution 1:1000, the tincture of chlorid of iron, etc.) and implements (post-nasal

Styptics.

tampon, artery forceps, sponge holder and stypic gauze—which can be used to exert direct pressure upon the bleeding spot; actual cautery, etc.), as will enable him to promptly check profuse hemorrhage.

RETROPHARYNGEAL ABSCESS

(Retropharyngeal Lymphadenitis).

Lymphadenitis.

Tuberculosis.

Retropharyngeal abscess is a disease of early infancy when the retropharyngeal lymph-nodes are in a state of highest development. It usually begins as retropharyngeal lymphadenitis, most frequently the result of infection by offensive nasopharyngeal discharges. More rarely it is due to spondylitis of the cervical vertebræ or occurs as a metastatic abscess or in consequence of trauma. Not all cases of lymphadenitis undergo suppuration; on the contrary, quite many retrogress and escape attention. Hence the apparent rarity of retropharyngeal disease. Some cases undergo suppuration and break spontaneously, and others run a rather latent course, and when seen by the physician present fully developed abscesses. Digital examination of the throat usually

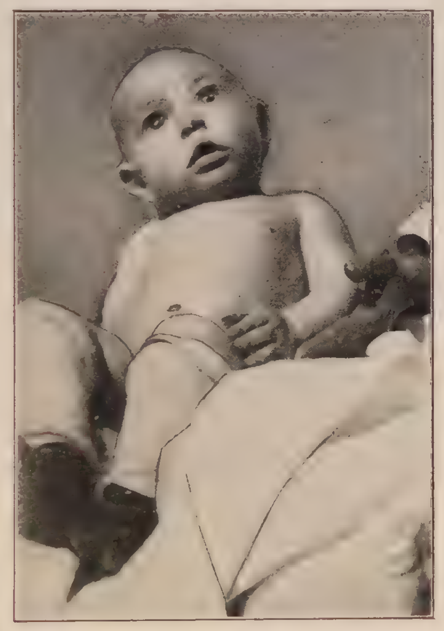

Fig. 70.—Retropharyngeal Abscess. Note characteristic attitude of head—"Pseudotorticollis." (*Sheffield.*)

reveals a round or oval fluctuating mass the size of a pigeon's egg, in the median line of the pharynx, and more rarely laterally on a line with the velum palati or somewhat below it. In the advanced stage the abscess may be recognized as a bluish-red tumor on ordinary inspection of the pharynx.

Round mass in pharynx.

The symptoms vary with the size of the tumor. In marked cases they consist of dysphagia, snoring respiration, especially during sleep, muffled voice and, with progressive growth of the swelling, dyspnea and attacks of asphyxia. Where deglutition is very painful there is also sympathetic *pseudo*-torticollis. Occasionally the submaxillary, parotid and other neighboring glands are involved, and in spontaneous rupture of the abscess metastatic abscesses are apt to develop in the supraclavicular fossa, mediastinum, and lungs.

Dysphagia.

Snoring.

Pseudotorticollis.

Treatment.—Early opening of the abscess is therefore imperative. This is best accomplished by gently perforating it by means of a pointed artery clamp and widening the puncture by opening the clamp. As soon as the perforation is made the child's head should be promptly bent forward to prevent the pus from entering the larynx (danger of asphyxia, aspiration pneumonia, etc.) and the nose and throat cleared of blood, pus and mucus.

Evacuation of pus.

In multiple communicating abscesses with palpable involvement of the adjacent glands, the operation is preferably performed (with a knife) from the outside so as to afford thorough drainage.

Relief from the symptoms is very prompt after evacuation of the pus. Rapid recovery, however, occurs only in primary streptococcic or staphylococcic abscesses; in metastatic and tuberculous abscesses the disease proceeds a protracted course, the prognosis depending upon the original disease and the age and vitality of the patient.

OTITIS MEDIA.

The gravest feature of nasopharyngeal affections, be they primary or secondary, is their great tendency to ear complications. The nasopharynx and ear being in direct communication through the Eustachian tube, infectious material can readily travel from the nose and throat to the middle ear and transfer the disease from one locality to the other. Hence the frequency of ear disease in rhinitis, adenoids, divers exanthematous affections, influenza, etc. Only a small percentage of cases of otitis

Secondarily to nasopharyngitis.

Traumatism.

"Dry" catarrh.

media are contracted through traumatism or extension of an inflammation from the external auditory meatus, and, in infants, middle-ear disease with masked symptoms is occasionally observed in connection with wasting diseases (*e.g.*, tuberculosis, marasmus, syphilis).

Earache.

Injection of drum.

Purulent discharge.

Mastoiditis.

Marked swelling.

Constitutional symptoms.

Involvement of sinus; meninges.

The infection may remain limited to the Eustachian tube (*catarrh of the Eustachian canal*), and give rise to very few and mild symptoms. The child may complain of earache for a day or two, perhaps, wake up at night with a crying spell, but get immediate and usually permanent relief after application of heat or some "ear drops." Sometimes the pain may return and get much more intense, and examination of the drum would show injection of the drum or, perhaps, a slight mucopurulent discharge indicating spontaneous rupture of the membrane. The discharge may continue for a few days or weeks and disappear without further ado. In another group of cases, due to greater virulence of the infective material or possibly neglect, the inflammatory process pursues a more violent course (*otitis media purulenta*). The temperature rises, the earache is very intense, the child is very restless, cries almost incessantly, rubs or strikes the ear with its hands, and as the symptoms persist there may be vomiting and cerebral irritation up to convulsions. If the pus is not evacuated, we soon find that it eats its way into the deeper structures, leading either to an acute or chronic involvement of the bone (*mastoiditis*). In severe infections this stage of the disease is often reached within a few days. The aforementioned constitutional symptoms are greatly exaggerated. The local signs, in addition to intense earache, deafness, headache and marked congestion of the drum, also are augmented by tenderness over the mastoid process and by swelling of the tissues covering the bone, extending downward along the entire side of the neck and forward to the retromaxillary fossa, pushing the auricle forward. The upper and posterior walls of the meatus are more or less swollen and the drum is highly inflamed, bulging and irregular in contour. The further course of the affection depends greatly upon the mode of treatment. If the inflammatory process is allowed to continue the pus may find its way either externally, somewhere along the side of the neck, into the throat (retropharyngeal abscess) or, in malignant cases, into the lateral sinus (*phlebitis, thrombosis*), or the middle fossa of the skull (*meningitis, purulent encephalitis*). The same grave condition is sometimes observed in otitis pursuing a very slow course—months or years. In these cases it is usually found that the patient is suffering from recurrent attacks of earache with or without profuse purulent discharge, more or less severe headache, dizziness, occasional rise of temperature, tenderness over the mastoid process, and, toward the end, loss of weight, anorexia, persistent headache and repeated vomiting.

Cerebral abscess.

The disease having reached this deplorable stage one is very rarely apt to err in the diagnosis. A question may arise as to whether the meningeal symptoms are secondary to otitis or to some other affection (*e.g.*, pneumonia, sepsis), or primary in character. A history of ear disease and the presence of local ear symptoms (discharge; inflammation of the drum, etc.) at once point to its true nature. Neither is there any difficulty in diagnosing otitis media purulenta with acute symptoms. The diagnosis, however, is not so easy in cases with an insidious course. It is especially difficult when the ear symptoms are masked by manifestations of the primary affection (*e.g.*, influenza), but an otoscopic examination almost invariably clears up the diagnosis, and should always be resorted to whenever inexplicable pain or temperature prevails. Only very recently I had occasion to find double otitis in a boy 14 months old who, for three weeks, was treated by a prominent clinician for "central pneumonia." Mild cases of middle ear disease may be mistaken for otitis externa. In this affection, however, the local signs are limited to the external auditory canal (redness and narrowing of the meatus without involvement of the drum). Similarly middle ear disease may be confounded with furunculosis or foreign bodies in the auditory meatus, but these can readily be eliminated by an otoscopic examination showing the seat of the lesion. Occasionally an abscess in the external canal burrowing itself through the cartilaginous portion of the canal in back of the ear may be mistaken for mastoid abscess; in such cases constitutional symptoms and inflammation of the drum are absent and the abscess is superficial and communicating with the swelling in the external canal.

Differentiation from "central" pneumonia.

Differentiation from external ear disease.

Bearing in mind the great tendency of nasopharyngeal affections to lead to ear disease, and the latter to become a source of everlasting misery and death, it is self-evident that all precautions should be taken to prevent the causes and their dreadful results. During the course of acute febrile, especially exanthematous dis-

Attention to nasopharynx.

Removal of adenoids.

Tapping

Paracentesis.

"Dry" treatment.

Icebag.

Radical operation.

eases, the nasopharynx should receive especial attention in the way of careful, gentle cleansing. Warm salt water or albolene should be instilled into the nose twice daily, preferably with a spoon or dropper, lest forcible syringing may drive the discharge from the nasopharynx into the Eustachian tube. Hypertrophied tonsils and adenoids should be removed and chronic nasopharyngeal catarrh treated with appropriate remedies. The instillations should also be continued after the appearance of ear symptoms, and as long as the membrane is intact syringing of the ear with warm boracic acid solution will prove beneficial. If the otitis continues and the drum does not rupture spontaneously, free paracentesis should be performed without delay, to allow the pus to escape. The mode of after-treatment is still subject to controversy, several prominent otologists preferring the "dry" method (drying of the external auditory canal several times a day and loosely packing with absorbent gauze) to repeated syringing. Where the discharge continues instillation of a few drops of a 2 per cent. solution of nitrate of silver, or in very chronic cases cauterization of the tympanum with trichloracetic acid will be found to act splendidly. If sensitiveness over the mastoid is detected and the constitutional symptoms show that disease is rapidly growing worse, an attempt should be made to arrest its progress by a new paracentesis, icebags and leeches and, if improvement does not set in early, there is nothing else left but immediately to proceed with opening of the mastoid process with a chisel to prevent the pus invading the sinus, meninges or brain substance. In the majority of instances a radical mastoid operation is a life-saving procedure. Unfortunately this operation is not rarely undertaken either too late or on a patient in a state of very low vitality from the baleful effects of the primary disease, so that the results are not always very gratifying. It is questionable whether operative interference is to be advised after the disease has spread to the meninges or brain. The recoveries in these cases are certainly very few and far between.

LARYNGITIS ACUTA.

Catarrhal Laryngitis; Spasmodic or False Croup; Laryngitis Stridula; Membranous, Non-diphtheritic Croup.

Acute primary, idiopathic laryngitis is comparatively rare in children, except as the result of the traumatic action of strong gases, vapors, fluids or excessive heat. On the other hand, laryngitis quite frequently occurs in conjunction with divers acute exanthematous diseases, especially measles and influenza, often follows attacks of rhinitis, pharyngitis, tonsillitis and esophagitis, and may develop in connection with intra- and extra-laryngeal growths. This so-called secondary laryngitis affects children principally of from two to ten years of age.

Secondary.

The severity of the symptoms is often by far out of proportion to that of the underlying anatomic lesion. Thus, simple hyperemia of only a small portion of the laryngeal mucous membrane not rarely gives rise to marked symptoms of suffocation.

Several forms of laryngitis are noted in practice:—

1. Catarrhal Laryngitis.—The child complains of sore throat and sensitiveness of the larynx to pressure. The cough is dry, short, and barking; the voice husky or only slightly muffled. Respiration is normal; fever is absent or slight. Expectoration is at first slight and of a mucous nature, later more profuse and mucopurulent. The attack lasts about a week.

Barking cough.

Occasionally, especially in neglected cases or in those suffering from affections of the nasopharynx, the laryngitis may pursue a chronic course with a tendency to permanent alteration of the voice. In this event laryngoscopic examination usually reveals a moderate hyperemia of the laryngeal mucous membrane, and in some cases slight erosions.

2. Spasmodic Laryngitis (Laryngitis Stridula, False Croup).—It develops, either very suddenly or after a few days' illness, with catarrhal laryngitis or nasopharyngitis. Sudden attacks usually occur in children under eight years of age, more frequently boys than girls. After retiring apparently healthy and sleeping fairly well until about midnight (this may also happen during the day after prolonged sleep, when the nasopharyngeal or laryngeal secretion desiccates and gives rise to irritation of the larynx, and possibly edema of the subchordal tissue) the child wakes up with a harsh, croupy cough, interrupted by deep inspiratory stridor. The child looks frightened and anxiously gasps for air, its face is flushed and bathed in perspiration, its eyes stare and its lips are cyanosed, and the whole clinical picture is very alarming. The dyspnea usually passes off in a few minutes but may last hours with slight remissions and gradual improvement. Ordinarily the child is well again in the morning except for a simple mild laryngitis which may subside in two to ten days or give rise to renewals of the

Sudden attacks of croup.

Intense dyspnea.

attack for a few successive nights. Sometimes the paroxysm may be so severe as to require intubation or tracheotomy for immediate relief. Spasmodic croup occasionally forms the beginning of pertussis, measles, influenza or membranous, non-diphtheritic croup.

Ascending.

Descending.

Dyspnea, cyanosis, eventually asphyxia.

3. **Membranous, Non-diphtheritic Laryngitis.**—In the beginning the symptoms are those of simple laryngitis. Very soon, however, the catarrh is increased in intensity. The cough becomes harsher and more croupy, the voice hoarse (sometimes aphonia), inspiration prolonged and expiration noisy. It may begin also with bronchial catarrh and become suddenly complicated by fibrinous tracheolaryngitis—*ascending croup*—reach a very high degree of intensity, become more severe from hour to hour, and threaten suffocation, if not immediately relieved by intubation or tracheotomy. The aspect is still worse when the croupous inflammation descends into the bronchi—*bronchial croup*. In this condition the patient may cough up white reticulated shreds (which float in water) or complete cylinders with dichotomic ramifications or multiple dendritic branchings. The prognosis in such cases is very grave. The pulse fails, the dyspnea and cyanosis increase, the patients fall into a state of sopor and die from collapse. Not infrequently fatal brain symptoms occur as a result of passive venous congestion in the brain and transudation in the ventricles. The course and termination of the disease, however, is not always so bad, and quite a number of uncomplicated (sometimes complicated by bronchopneumonia) cases recover without much ado.

Differential diagnosis.

This non-diphtheritic form of laryngitis is often mistaken for diphtheritic membranous laryngitis, but a diagnosis can in the majority of cases be made with the aid of the following differential points:—

MEMBRANOUS DIPHTHERITIC LARYNGITIS.	MEMBRANOUS NON-DIPHTHERITIC LARYNGITIS.
Diphtheria bacilli present.	Absent.
Distinctly contagious, giving also a history of contagion.	Not contagious.
Early enlargement of the submaxillary glands.	Submaxillary glands, as a rule, not involved.
Diphtheritic patches are found, as a rule, on the fauces and posterior pharyngeal wall.	The fauces may be covered with a mucous exudation, which can easily be wiped off.
Albuminuria usually present.	Absent.

Treatment.—Mild cases do nicely on very simple therapeutic measures such as rest in bed, hot baths, hot drinks (tea, Salicylates.

lemonade, milk and seltzer), Priessnitz's compresses or turpentine and camphorated oil to the neck and a few doses of sodium salicylate internally to relieve the sore throat and to stimulate diaphoresis.

Inhalations.

Should there be any tendency for desiccation of the laryngeal secretion, softening of the same should be endeavored by means of expectorants, steam inhalations and emetics. In the majority of instances this mode of treatment prevents the occurrence of attacks of spasmodic laryngitis.

Sedatives.

℞ Vini ipecacuanhæ	ʒss.	2.00
Syr. scillæ comp.	ʒj.	4.00
Syr. senegæ	ʒij.	8.00
Codeinæ sulph.	gr. ss.	0.03
Mist. glycyrrhizæ comp.	q. s. ad f℥ij.	60.00

M. Sig.: One teaspoonful every two to four hours for a child 3 years old.

℞ Eucalyptol	ʒj.	4.00
Tinct. benzoini comp.	℥ij.	60.00

M. Sig.: One teaspoonful in a pint of hot water for inhalation.

Emetics.

Sudden paroxysms of false croup are best remedied by prompt emesis, a hot mustard bath (see page 106), a hypodermatic injection of morphine and atropine, counterirritation by a strong sinapism and, if the cyanosis increases notwithstanding, intubation or tracheotomy.

Antispasmodics.
Intubation.
Antitoxin.

The management of membranous non-diphtheritic croup is frequently quite a difficult proposition. Hence, the importance of its prevention by early attention to catarrhal laryngitis. Steam inhalation (see above) and emesis are useful remedies, and inhalation of amyl nitrite or chloroform is often effective to relieve threatening dyspnea. Severe cases call for early intubation or tracheotomy. Recurrent laryngeal spasm sometimes yields to spraying of the larynx with 2 per cent. sol. of cocaine. As diphtheria antitoxin carefully administered is a safe remedy, it is always advisable to resort to it, although bacteriologic examination of the pseudomembrane fails to reveal the diphtheria bacillus.

Prophylaxis.—Removal of local causes, such as adenoids and large tonsils; change of air; tonics, especially cod-liver oil.

LARYNGITIS CHRONICA.

Chronic laryngitis may follow repeated attacks of acute catarrhal or diphtheritic laryngitis or develop slowly by extension

Trache

of inflammation from the neighboring structures. Overexertion of the voice and excessive smoking in boys are occasionally causes.

Inflammatory symptoms.

Laryngoscopic examination shows hyperemia and swelling of the mucous membrane of the larynx which vary in extent with the duration of the affection. The mucous membrane is sometimes covered with granulations and in severe cases shows more or less superficial ulceration. There is a moderate secretion of mucus and pus which has a tendency to desiccate, and gives the sensation of a foreign body in the throat. The cough is usually insignificant, occasionally, however, troublesome, harsh and barking, especially at night.

Resembles syphilitic and tuberculous laryngitis.

Diagnosis.—Although syphilis and tuberculosis of the throat are comparatively rare in children, their presence should always be suspected and looked for in obstinate laryngitis. The following differential points are helpful in the diagnosis:—

	SIMPLE LARYNGITIS	SYPHILITIC		TUBERCULOUS
		SECONDARY	TERTIARY	
Lesion	Hyperemia, slight thickening, erosion of mucous membrane, rarely slight ulceration	Mottled hyperemia, superficial ulceration	Deep, angry ulcers, cicatrices, stenosis	Anemia, grayish color, solid thickening, worm-eaten ulcers
Expectoration.	Free from tubercle bacilli	Spirochetes	The same	Bacilli present
Deglutition ...	Usually painless	Normal	Difficult	Very painful
Cough.........	Dry or moist, painless	Slight hacking	Infrequent	Severe, as a rule
Respiration ...	Normal	Unaltered	Embarrassed with stenosis	Early acceleration
Voice	Variable	Hoarse, nasal	Raucous, husky	Partial or complete aphonia
Complications.	Nasopharynx; general health unaffected	Syphilitic lesions elsewhere	The same	Involvement of lungs, emaciation

Treatment.—Attention to existing causes, especially adenoids and enlarged tonsils if present; local application, three times a week, of nitrate of silver (1 per cent. to 2 per cent.), glycerate of tannin (10 per cent.), or chlorid of zinc (2 per cent. to 4 per cent.); steam inhalations (see page 256); cleansing of the nose and throat, three times a day, with Dobell's solution, and the like, will very promptly effect a cure, provided the laryngeal affection is not based on some grave constitutional affection, or benign (papilloma) or malignant growths. Rest to the voice is of material benefit. In very protracted cases change of air and

Nitrate of silver.

Removal of growths.

constitutional treatment. Faradization of the larynx is often very serviceable to relieve aphonia.

℞ Codeinæ sulph.	gr. ss	0.03
Creosoti carbon.	ʒss	2.
Syr. acaciæ	q. s. ad fʒij	60.

M. Sig.: One teaspoonful every three hours for a child 6 years old.

ŒDEMA GLOTTIDIS.

Active

Passive or secondary.

Edema of the larynx occurs in two forms: Active (inflammatory, phlegmonous), and passive (serous). Inflammatory edema may be primary, usually traumatic (*e.g.*, scalds or burns), or secondary, as a result of extension of inflammation from neighboring structures. Passive edema is usually observed in connection with grave kidney and heart diseases—often long before dropsy is manifested in any other part of the body—and secondarily to pressure on the larynx by swellings or growths.

Pathologic findings.

Anatomically edema of the larynx consists of a yellowish-white or reddish tumefaction—a serous, seropurulent or sanguinolent transudation into the submucosa—involving the upper portions of the larynx, the epiglottis, the aryepiglottic folds, the false (rarely the true) vocal cords, and the mucous membrane of the arytenoid cartilages.

These local changes can readily be detected by inspection of the larynx, often without the mirror, by simply depressing the tongue and pulling it forward, and by digital examination.

Dyspnea.

The result of such swelling of the laryngeal tissues is quite obvious, namely, interference with normal respiration. The dyspnea is at first paroxysmal, and, if the edema is not very marked, only moderately severe. The poor little patient hacks and coughs, in vain trying to clear the throat. If the edema advances, the dyspnea becomes extreme; symptoms of asphyxia set in which, if not promptly relieved, lead to a fatal issue.

Œdema glottidis should not be mistaken for spasmodic croup!

Sedatives.

Tracheotomy.

Treatment. — Partial edema may be reduced by icebags to the neck, swallowing of ice, local application of suprarenal extract solution (1:1000) and morphine and pilocarpine hypodermatically. In severe cases tracheotomy should be resorted to in addition to the mode of treatment just outlined. Recurrence of an attack should be prevented by prompt attention to the etiologic factors.

LARYNGEAL TUMORS.

Neoplasms of the larynx are very rarely seen in children. This is especially true of malignant growths. *Papillomata* are not quite so rare, and are sometimes congenital. Their usual seat is at the true vocal cords, and if of considerable size they give rise to obstinate, severe cough, hoarseness, dyspnea and attacks of asphyxia. These symptoms develop however, gradually, and sometimes disappear spontaneously owing to retrograde metamorphosis of the tumor. Recurrences are frequent. Laryngeal neoplasms may be confounded with adenoids, retropharyngeal abscess and croup, but the diagnosis can readily be made by laryngoscopic examination. Operative treatment should be instituted only in cases presenting troublesome symptoms. Endolaryngeal removal of the growth is the procedure of choice. Tracheotomy, in threatening asphyxia.

Papillomata.

Differentiation from adenoids, retropharyngeal abscess, and croup.

FOREIGN BODIES IN THE LARYNX.

Various articles of food, little playthings, buttons, needles, ascarides, etc., may find their way into the larynx. Small foreign bodies are usually expelled by the attacks of forcible coughing. Larger non-impacted articles may be removed by an extubator or similar forceps after cocainizing the upper part of the larynx. Foreign bodies firmly impacted in the larynx should be removed under anesthesia through the tracheotomy incision. In threatening asphyxia tracheotomy should be performed immediately irrespective of subsequent procedures. To reduce hyperemia, ice externally and internally. Local antiphlogosis (Lugol's solution, 1 per cent. nitrate of silver) after removal of the foreign body. Anodynes for the relief of pain and irritability. (For removal of ascarides see page 226.)

Tracheotomy in threatening asphyxia.

DISEASES OF THE LUNGS AND PLEURA.

BRONCHITIS AND BRONCHOPNEUMONIA.

Tracheobronchitis; Capillary Bronchitis; Lobular Pneumonia.

Bronchopneumonia in children is usually secondary in nature (forming a complication of divers acute and chronic diseases) and is generally preceded by or associated with a catarrhal inflammation of the mucous membrane of the trachea and bronchi. As the tracheobronchitis advances the inflammation spreads to the fine bronchioles—capillary bronchitis—and, finally, to the pulmonary alveoli—lobular- or broncho-pneumonia. In the latter affection the consolidation is irregularly distributed, sometimes over the entire lung, in variously sized patches. On section the affected lobules present quite a smooth surface of bluish-red color, and contain a mucosanguinolent fluid. When placed in water they sink to the bottom. In cases of long standing atelectasis, emphysema and caseation are common complications.

Irregular distribution of lesion.

Short, dry cough.

In *tracheitis* the cough is short, dry and harsh, becoming longer and softer as the inflammation extends to the bronchi. Respiration is but little embarrassed, the temperature is normal or slightly elevated and the general health corresponds with the underlying condition. The onset of *bronchitis,* on the other hand, is signalized by a rise of temperature of from two to three degrees, sometimes vomiting and marked restlessness. The cough is frequent and painful, breathing is accelerated and somewhat difficult, and auscultation reveals a great number of large, harsh and moist râles and sibilant rhonchi which are transmitted over the entire chest wall and give rise to the characteristic wheezing and whistling which are readily heard at some distance from the patient and felt by the palpating hand. This "rattling of the chest" usually diminishes in intensity or disappears temporarily after forcible coughing.

Large râles.

Wheezing.

Under suitable treatment the tendency of primary tracheobronchitis is toward gradual evanescence. After a few days the disease assumes a milder course; the cough becomes looser and less frequent; the breathing slower and less noisy; the general condition rapidly improves, and recovery is often complete within from seven to fourteen days.

Often chronic course.

Not infrequently, especially in secondary bronchitis, where the primary etiologic factors remain active, or in neglected cases, the catarrh pursues a protracted course (chronic tracheobronchitis); aggravation of the condition alternates with amelioration; the child continues to hack or cough for weeks or months, presents large and moist râles over different portions of the chest, but may otherwise remain free from any constitutional symptoms. In a small number of cases chronic bronchitis forms a precursor of tuberculosis of the bronchial glands or lungs.

Tendency to tuberculosis.

In young and delicate children tracheobronchitis is always fraught with the danger of terminating into capillary bronchitis or bronchopneumonia. Indeed transition of the inflammation from the large bronchi to the fine bronchioles (*bronchiolitis, capillary bronchitis*) and the lung tissue (*bronchopneumonia* or *lobular pneumonia*) not rarely proceeds insidiously, and may exist for some time, especially in the lower lobes, before being detected.

Transition into capillary bronchitis or bronchopneumonia.

As a rule, extension of the pulmonary inflammatory process is associated with sudden rise of temperature (up to 105° F.) with its concomitant symptoms, and increased frequency of respiration. The cough becomes dry, short and very painful. The nostrils dilate and contract. The eyes are dull. The face is pale, cyanotic, and often covered with perspiration. No mathematical distinguishing line can be drawn between the symptoms and physical signs of capillary bronchitis and catarrhal pneumonia, except, perhaps, that in capillary bronchitis the pulmonary lesions are more diffuse (the whole bronchial tree may suddenly become involved) while in lobular pneumonia more local. As the disease advances the local pneumonic foci gradually multiply, become larger and more confluent, and coalesce in extensive masses. Then, and often not until then, can dullness be demonstrated on percussion. Where the patch is small the percussion note may be normal or even tympanitic. Inspection discloses retraction of the lower ribs during breathing. Auscultation elicits accentuation of the expiratory sound, bronchial breathing, bronchophony over the dull portions and fine crepitation, in addition to large, soft and sonorous râles, distributed over different parts of the lungs, especially over both sides of the spine, and along the axillary lines.

Rise of temperature.

Rapid breathing.

Confluence of lesions.

Dullness.

Bronchial breathing.

Lobular pneumonia being usually a secondary affection (primary pneumococcic bronchopneumonia may, like lobar pneumonia, end by crisis) runs a very protracted course, from two to six weeks or longer. This is often due to repeated extension of the inflammatory process, sometimes with disappearance of the original focus. This accounts also for the apparent improvement and relapse. Under the circumstances the wear and tear upon the child's constitution is very great, especially since with persistent anorexia tissue repair is at complete abeyance.

The heart's action grows weaker; the power to cough diminishes, notwithstanding exaggeration of the physical signs; breathing becomes more difficult, and the pulmonary circulation more and more obstructed. The child finally succumbs to autoinfection and cardiac exhaustion (the overdistended right heart being

Dyspnea.

Heart weakness.

unable to propel its contents, not rarely preceded by attacks of suffocation, coma, and convulsions.

Grave prognosis.

The prognosis is not always so grave. In some cases, especially in children whose constitution has not previously been undermined by wasting diseases, defervescence occurs after a week or so, the dullness diminishes, the cough loosens, sleep becomes more restful, respiration less painful, the appetite returns, and if not interrupted by complications, gradual recovery ensues within a few weeks.

On the other hand, fatal termination after a few days' sickness is not at all rare. This is more apt to occur in primary, pneumococcic bronchopneumonia from an overwhelming toxic effect upon the heart muscle and the cerebrum (meningitis, encephalitis).

Complications.

Pyothorax, miliary tuberculosis, gastroenteritis, stomatitis, more rarely otitis, pleuritis, and gangrene of the lungs, form the principal complications. Empyema, tuberculosis and pulmonary gangrene are usually found only in cases of bronchopneumonia with delayed resolution, as a result of caseation and liquefaction of the unabsorbed inflammatory products.

Spreads by continuity.

Treatment.—The management of bronchopneumonia in young children depends upon the underlying condition of the disease. Primary, pneumococcic, lobular involvement, like lobar pneumonia, usually proceeds a self-limited course and is little influenced by therapeutic measures. On the other hand, secondary catarrhal pneumonia spreads by continuity and may often be arrested in its inception by early and energetic treatment. This is true particularly of bronchopneumonia supervening tracheobronchitis—as already alluded to, in the beginning, a simple local catarrh, readily amenable to prompt attention.

Energetic treatment.

As the initial symptoms of bronchitis and bronchopneumonia are not always easily distinguishable, and as the success of our treatment invariably depends upon its promptitude, it is good practice to err in the direction of judicious overtreatment rather than in that of irresolute undertreatment, and to at once proceed with active therapeutic measures in tracheobronchitis and bronchopneumonia alike.

The treatment consists of induction of free perspiration, enhancement of expectoration, allaying nerve irritability and pain, and maintenance of the patient's strength. Seeing the patient in the early stage of the disease we direct the administra-

Hot mustard bath.

tion of a hot mustard bath of about three minutes' duration and the application of a poultice consisting of the following ingredients: Five parts each of flaxseed-meal and camphorated oil, one to two parts of mustard, and a sufficient quantity of *boiling* water to make a thick paste by thorough stirring. This mass is spread on thin gauze or paper (two layers) and applied snugly to the chest and back. The child is then wrapped in an oiled-silk jacket, lined with absorbent cotton, and in a blanket, which, with the hyperpyrexia of the body, maintains the heat of the poultices, so that renewal is required but three or four times in twenty-four hours. This poultice has special advantages over any other in use. As just mentioned, it requires but occasional changing, thus saving time and labor and avoiding unnecessary exposure of and annoyance to the patient. The mustard and camphor act as mild counterirritants, and after some time bring the blood to the surface, thus relieving the pulmonary engorgement. Furthermore, the skin over the chest and back does not become "soggy and sodden," or "water-logged" from the use of this poultice as is apt to occur from prolonged application of ordinary flaxseed poultices.

Local heat.

In conjunction with the external treatment the patient receives also a few doses of sweet spirits of niter and liquor ammonium acetate which act very kindly both as diaphoretics and stimulants.

Diuresis and diaphoresis.

The beneficial results derived from this mode of treatment are manifest within a few hours. The suffering infant who but a short time before had been on the verge of collapse—moaning, tossing and twitching from pain and distress, gasping and panting for a free breath of air—now lies peacefully enjoying calm repose and healthful sleep, ready and apparently able to battle for a new lease of life. The system having been greatly relieved of its toxemia by means of the free perspiration, the disease now usually assumes a much milder course. Indeed, it is not at all uncommon to see a severe attack of tracheobronchitis to end then and there, and that of bronchopneumonia to resolve itself into simple bronchitis.

Effects of treatment.

The enthusiasm just expressed applies, of course, only to such cases as are ordinarily met as a result of a "cold." This treatment is surely no panacea for respiratory embarrassment complicating grave affections of other bodily organs, *e.g.*, heart or kidney. Here symptomatic medication is in order—at best an unthankful task.

To enhance free expectoration we resort to the following expectorant mixture:—

℞ Ammonii carbon.	gr. xvj	1
Vini ipecacuanhæ	ʒss	2
Syr. scillæ comp.	ʒj	4
Syr. senegæ	ʒj	4
Syr. Tolutani	ʒiv	15
Aquæ destil. q. s. ad	℥ij	60

M. Sig.: One teaspoonful every two or four hours for a child two years old.

Anodynes.

To this we may add a small quantity of the camphorated tincture of opium (gtt. 2 to 5) or codeine sulphate (gr. 1/60) for the relief of pain and to allay nerve irritation. For the latter purpose an ice-bag to the head or sodium bromid internally often does well.

Expectorants.

We cannot pass this question without expressing our disapproval of the absurd criticism often heard as to the use of expectorants. When a little infant is tormented almost to death by an incessant, dry, hacking cough and the painful phenomena associated with it, it is no empiricism to administer an expectorant mixture which helps nature to rid the lungs of effete material (which more or less obstructs respiration and causes autoinfection by systemic absorption) and permits the patient to refresh upon a brief period of rest and sleep.

Stimulants.

Camphor.

The maintenance of the child's strength is most essential to the successful management of bronchopneumonia. The heart should be looked after from the very inception of the disease. For be it remembered that death in pneumonia is due to heart failure and not to pulmonary insufficiency. In the early stages of the disease we rely principally upon strychnin (gr. 1/100 to 1/50), but as the circulatory and respiratory difficulties increase we do not hesitate to administer camphor (gtt. x of a 15 per cent. sterilized camphorated oil) hypodermatically and digitalis and strophanthus by mouth, as indications demand.

Every effort should be made to replenish the vital body fluids consumed during the active febrile process by suitable nourishment such as water, milk, beef-tea, broths, fruit-juice, etc. (See also "Pneumonia.")

When called upon to treat a case of bronchopneumonia of several days' or weeks' duration that has failed to respond to active treatment, our efforts should be directed toward the prevention of pyothorax or tuberculous infiltration of the lungs.

Fresh air.

A great deal can be accomplished by an ample supply of fresh air, the iodids, creosote and essential oils by mouth and inhalation. (See also "Chronic Pneumonia.")

Whenever possible, the child should be removed to the country (seashore or mountains), and, weather permitting, kept outdoors most of the time.

Iodids.

The iodids will be found very useful to hasten resolution (preferably in the form of sodium iodid gr. ½, t. i. d. for a child one year old). We usually recommend its administration from the sixth day of the disease until resolution has been established, and then continue with the syrup of the iodid of iron and the syrup hypophosphite compound, which act both as an efficient alterative and tonic.

Creosote.

Creosote is indicated in all stages of the disease. It should be prescribed in small doses several times a day. The tent made of bed-sheets hung around the bed and moistened with creosote, oil of eucalyptus and the like is of service, especially in tracheobronchitis.

LOBAR PNEUMONIA.

Croupous or Fibrinous Pneumonia, Pneumonitis.

Acute lobar pneumonia is an acute, specific, inflammatory affection of the lung tissue arising as a result of invasion by the encapsulated diplococcus of Fraenkel-Weichselbaum. It may occur as an independent process or in connection with other diseases, *e.g.*, influenza, measles, diphtheria, scarlet or typhoid fever, etc. It is communicable and occurs occasionally in epidemic form.

Three stages.

Genuine pneumonia in children, as in adults, is characterized by three pathologic stages: Engorgement, red hepatization or consolidation, and gray hepatization, followed by resolution or purulent infiltration. The pleura is almost invariably implicated.

Sudden onset.

Chill.

Primary lobar pneumonia usually ushers in suddenly, often after exposure to cold or wet, with a chill, vomiting, high temperature, and more or less marked dyspnea. The initial symptoms are frequently misleading. They may consist of vomiting, diarrhea, pain in the abdomen, nosebleed, and prostration, suggesting the beginning of typhoid, or convulsions and vomiting may predominate justifying the diagnosis of meningitis. Where the pneumonic lesion is located centrally (*central pneumonia*), the physical signs, nay, even the cough, may be absent or slight,

Absence of cough in central pneumonia.

so that remittent fever is often thought of or even intermittent fever, if the temperature pursues an irregular course. Furthermore, there are also numerous abortive cases of pneumonia which terminate in a few days—often before the diagnosis has been established.

Fine crepitation.

Tubular breathing.

Dullness.

Of course, the majority of cases of pneumonia present typical physical signs and can be readily disclosed on careful examination. Auscultation reveals during the *first* and *third* stages fine crepitation at the edge of the consolidation, and during the *second* stage, distinct tubular breathing and bronchophony over the affected portion of the lung. In the first day or two of the disease the percussion sound is usually tympanitic, but as the pneumonia advances, first dullness and later flatness can readily be elicited, the experienced hand perceiving also a distinct increase of sense of resistance on percussion. Pectoral fremitus is ordinarily not sufficiently distinct in young children except when they cry aloud, which act should always be encouraged to facilitate the detection of the physical signs.

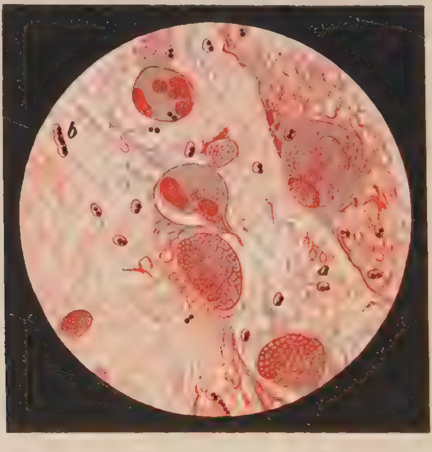

Fig. 71.—Diplococcus Pneumoniæ (Pneumococcus): (*a*) single diplococci; (*b*) the same in chains (Wolf's double stain). Leitz ocular I, oil immersion 1/12. (*Lenhartz* and *Brooks.*)

Self-limited.

Croupous pneumonia runs a self-limited course, between five and thirteen days, or longer, most frequently terminating by crisis, at a time when the disease is at its height. Until then, in the absence of unexpected complications, there is little change

Continued hyper-pyrexia.

Expiratory moan.

Crisis.

in the clinical picture of the affection. The fever remains high (104° to 105° F.), with slight morning remissions; the pulse- and respiration-ratio is greatly disturbed, from 1 to 3.5 to 1 to 2; the urine scanty, high colored, rich in salts (with diminution in chlorides) and occasionally in pepton and aceton; the cough is short, dry and painful; older children (rarely those under three years of age) expectorate rusty sputum; the face is flushed, the tip of the nose and lips are cyanotic; the tongue is coated, pasty, its tip red; the child is restless, sleepless, and tosses from side to side. As the crisis approaches and the circulation becomes more disturbed, the expiratory moan becomes louder and shorter, the cough more harassing, the breathing more superficial, the thirst more intense; the child lies helplessly (often rigid), usually on the affected side, in a state of apathy and exhaustion or in delirium. The change wrought by the establishment of the crisis is marvelous. Almost in an instant the heart's action calms down (the pulse is often irregular and very feeble); the breathing becomes slower, deeper; the tubular breathing-sounds are successively supplanted by crepitation, large and small soft râles, and normal vesicular respiration; the dullness on percussion gradually disap-

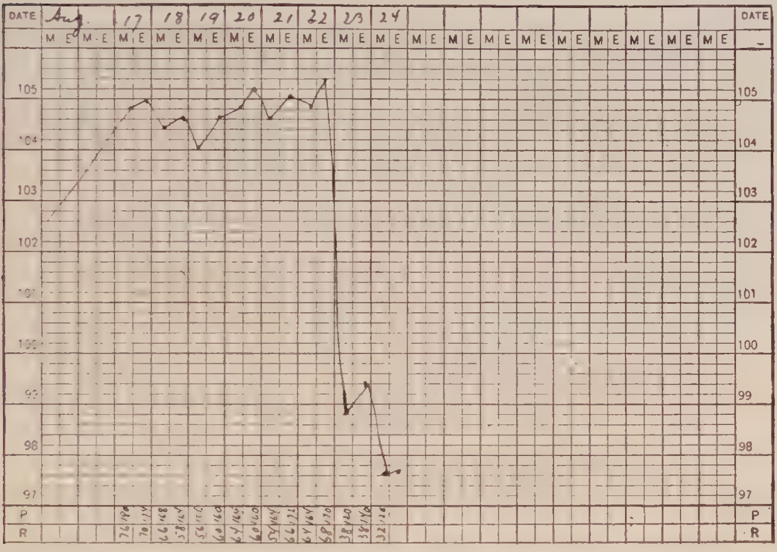

Fig. 72.—Fever Curve of Typical Lobar Pneumonia in a child 15 months old, ending by crisis. (*Sheffield.*)

pears; the cough diminishes, the temperature drops to normal or below it; the appetite improves and convalescence proceeds uninterruptedly, so that recovery is usually complete within a few days after the crisis.

Lysis. This favorable course is the rule. Exceptions are not rare. Not rarely pneumonia terminates by lysis. Defervescence may be tardy, the temperature reaching normal by two or three stages. Occasionally after a true crisis and apparently durable defervescence, recrudescence takes place; the temperature again rises and after a few days' persistence finally subsides by lysis. Wandering pneumonia. In some instances pneumonia runs a very protracted course; the inflammatory process "creeps" from lobe to lobe, and finally terminates in *unresolved, chronic* or the so-called *caseous* pneumonia. Complications. Quite frequently the pleura is markedly implicated, (*pleuropneumonia*), without or with an effusion into the pleura, in the latter event frequently resulting in *pyothorax*. Termination of pneumonia in pulmonary *gangrene* is rare.

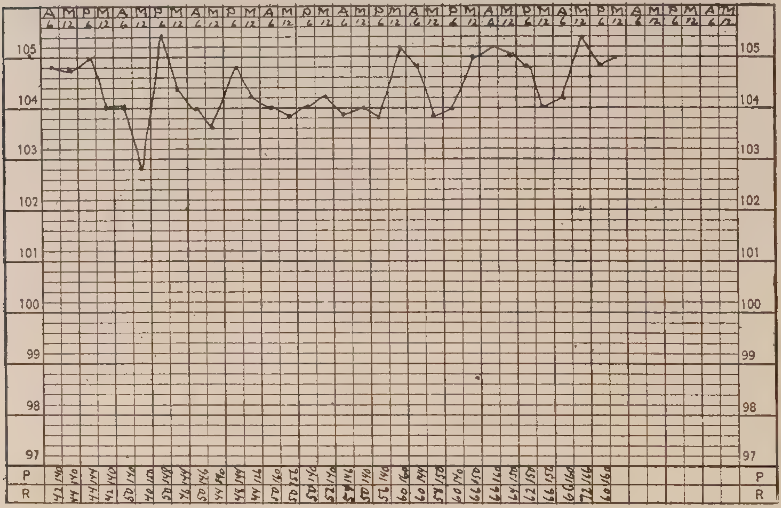

Fig. 73.—Fever Curve of Fatal Apex Pneumoniæ, with marked Cerebral Symptoms in a child 2 years old. (*Sheffield.*)

Primary pneumonia under suitable management offers quite a favorable prognosis. More serious are the cases in which the lung involvement is very extensive (*e.g., double pneumonia*) or located at the *apex*. Still less favorable is the pneumonia supervening other infectious and contagious diseases, especially if it

Complicated [illegible]

is preceded by a prolonged exhausting siege of the underlying affection. Complications substantially mar the prognosis as regards immediate and ultimate recovery, meningitis and pericarditis especially proving fatal. Acute nephritis usually ends in recovery. Gangrene, pyothorax, peritonitis and suppurative inflammation (pneumococcic metastases) of the bones and joints not rarely yield to early operative interference. Exceptionally fulminating attacks of pneumonia, with extremely high temperature, marked dyspnea, and very rapid and feeble pulse are met which prove fatal within two or three days. The relation between high leucocytosis and a favorable prognosis is still an open question.

Sequelæ.

Diagnosis.—In the initial stage lobar pneumonia may be confounded with lobular pneumonia, pleurisy, meningitis and intermittent fever.

Differentiation from: Broncho-pneumonia and pleurisy;

In the second stage, with pleurisy with effusion, and in the absence of cough (which often occurs when the bronchi are free), and the presence of abdominal pain and tympanites (the pleural pain is frequently erroneously referred to the abdomen; the tympanites is a result of intestinal fermentation, and swallowing of sputum and air) with peritonitis or appendicitis. Errors in diagnosis are prone to be made, especially in "central pneumonia" with masked physical signs.

Appendicitis;

In the third stage of the disease, with miliary tuberculosis and typhoid fever.

Miliary tuberculosis and typhoid fever;

Accidental supervention of angina and erythema (the latter often as a result of heat or rubefacients) may lead to the suspicion of scarlatina.

[illegible]

Scarlatina.

Bearing in mind the characteristic symptoms of the diseases for which pneumonia is apt to be mistaken, there ought to be no difficulty in eliminating most of them. The greatest difficulty is usually experienced in the differential diagnosis between acute lobar pneumonia and catarrhal pneumonia and acute miliary tuberculosis:—

ACUTE LOBAR PNEUMONIA.	CATARRHAL PNEUMONIA.
Generally a primary disease.	Secondary.
Onset sudden.	Gradual.
High regular fever.	Moderate and irregular.
Inflammatory process localized.	More diffuse.
Physical signs distinct.	Indistinct.
Termination by crisis the rule.	By lysis.

ACUTE LOBAR PNEUMONIA.	MILIARY TUBERCULOSIS.
Onset sudden and marked.	More gradual and masked.
Fever high and regular.	Very irregular.
Tuberculin test negative.	Positive.
Sputum contains pneumococci.	Tubercle bacilli.
Duration from one to two weeks with tendency to recovery.	From three to six weeks, ending fatally.

See also Pleurisy, page 278.

Communicable.

Pneumonia being a communicable affection it calls for all such hygienic precautions as are ordinarily employed for the prevention of other contagious and infectious diseases. As the contagium is carried by means of the sputum, the latter should be collected in separate receptacles, preferably small pieces of gauze, and destroyed.

The active treatment of pneumonia is essentially symptomatic—intended principally to make the patient comfortable and to maintain his power of resistance.

Fresh air.

Fresh air is the *sine qua non!* It purifies the respiratory tract, eases respiration, facilitates pulmonary circulation; hence, relieves and regulates the heart's action, reduces temperature, cheers the patient in those endless, wakeful hours, which are characteristic of pneumonia, and, last but not least, disinfects the sick-room, and thus prevents transmission of the disease to others and autoinfection of the patient.

Pure water.

Plenty of pure drinking water is the next most important requisite. This heavenly beverage should be given to the little patients *ad libitum,* unless temporarily contraindicated by uncontrollable vomiting, when only small quantities should be administered. Pure water cleanses the mouth and alimentary canal, which in children with pneumonia is usually infected by the large quantities of putrid sputum swallowed; it quenches the ever-present, agonizing thirst; stimulates expectoration and aids in the reduction of temperature. Moreover, at a time when anorexia is complete, little children, like fish, seem to subsist solely on water, and when the body fluids, desiccated by the burning heat, are at a very low ebb, nature seems to find in water a grateful auxiliary, to "turn the tide."

Enteroclysis.

It is very advantageous to have a standing order to employ daily a high intestinal irrigation to cleanse the bowels and to relieve the painful flatulence. Enteroclysis may be repeated a few times a day for the purpose of stimulating the action of the heart and kidneys.

Hydro-therapy.

Water should serve as the only antipyretic, when reduction of fever is indicated. As long as the temperature is below 103° F., no antipyresis is necessary. In higher temperatures, sponging, warm baths, cold packs, and, in older children, cold baths followed by brisk friction in accordance with the directions given under "Hydrotherapy," should be resorted to.

Occasionally we are called upon to relieve pain, allay the dry cough, subdue the nervous irritation and to support the heart's action.

Counter-irritation.

Pain in pneumonia is best relieved by local heat, either in the form of a flaxseed and mustard poultice (see page 263) or cloths immersed in warm mustard water and wrung out, and covered with oiled silk. These may be left in place for from ten to twenty minutes, and repeated twice or thrice a day or more often if the pain persists.

The same local remedies are also efficient to lessen the harassing cough.

Excessive nerve irritability is either the result of toxemia or hyperpyrexia, or both. If due to high fever the treatment is self-evident. As warm baths combine antipyretic and soothing qualities to the nerve system, they are admirably adapted for the purpose. In cerebral irritation caused by the bacterial toxins an effort should be made to eliminate the latter by colon irrigation, diuresis and hot baths. In the majority of instances, however, we have to have recourse to the bromids, chloral and similar remedies, especially when convulsions supervene and not rarely threaten the life of the patient.

Camphor and strychnine.

Cardiac debility setting in early is a very grave proposition. Camphor and strychnine in gradually increasing doses are best suited for the purpose. Whenever possible, stimulation should be employed hypodermatically, to obviate gastric disturbances. In severe cases nitroglycerin and digitalis are indicated. We should not exhaust all stimulants at once, but always keep one stimulant in reserve to have something to fall back on when an urgent necessity arises.

Iodids.

Protracted and unresolved pneumonias respond favorably to the iodids, which may in small doses be begun with about the fifth day of the disease. Iodism can readily be prevented by minute quantities of belladonna.

The problem of feeding pneumonia patients is a very difficult one. It is well to bear in mind that a filled stomach by upward

Feeding.

pressure greatly interferes with respiration and cardiac action. The nourishment should be very light and easily digestible, and given in very small quantities. It is remarkable how often apparently delicate infants withstand a very tedious and trying course of pneumonia with barely any food. Breast-fed babies when suffering from dyspnea should receive mother's milk from a spoon, since by overlapping the child's mouth and nose the breasts are very apt to cut off the little pure air supply the child is able to obtain.

The mouth and nasopharynx should be cleansed twice a day.

[illegible] salt water, boric acid 5%

CHRONIC PNEUMONIA.

Unresolved Pneumonia, Fibroid Pneumonia.

Pathology.

The mode of development of chronic or unresolved pneumonia has already been referred to when speaking of lobar and lobular pneumonia (*q. v.*). The lymph in the lungs degenerates into fibrous tissue and caseous matter, and the pulmonary interstitial connective tissue undergoes hypertrophy, leading to induration and contraction of the parenchyma, and bronchiectasis.

Tendency to tuberculosis.

After apparent termination of the pneumonia, the child continues to cough, fails to regain its strength, suffers from embarrassed respiration, and now and then exhibits rise of temperature. Examination of the chest reveals circumscribed areas (most frequently over the upper lobes) of dullness, bronchial breathing, bronchophony and large crepitant râles. In children with an undermined constitution or an hereditary tuberculous disposition this condition often gives rise to phthisis and early death. Stronger children, especially if the lesion is small, may after an indefinite period of suffering finally recover.

Change of air.

Treatment.—Early attention to the pneumonia, in its acute and chronic states, is of primary importance. Removal of the patient from stuffy unsanitary rooms, and allowing a free influx of pure air will do much to prevent the destructive tendencies of the infected foci. With the same object in view we must avoid administering drugs which suppress cough—nature's method of clearing the lungs of impurities. Early sojourn in the mountains and mild seashore resorts; nutritious food; breathing exercises; the internal administration of small doses of creosote and guaiacol and large doses of the syrup of the iodid of iron with cod-liver oil and malt will help to enhance a cure.

℞ Guaiacol carbon.	ʒss	2
Chocolate	ʒj	4

M. Ft. pulv. no. xv.
Sig.: One powder every four hours for a child 4 years old.

PLEURITIS
(Pleurisy).

Primary. Secondary.

The pleura, like other serous membranes, may be affected, *primarily* as a result of trauma, or invasion of pathogenic bacteria, such as the pneumococcus, streptococcus, the microbe of rheumatism, etc., or *secondarily* by extension of an inflammation from neighboring structures. Primary pleurisy is comparatively rare in young children. The secondary variety, however, is quite common in connection with pneumonia, tuberculosis, acute heart disease, and affections of the abdominal organs.

Pathology.

Pathologically pleuritis is characterized by congestion and roughness of either the parietal or visceral layer of the pleura or of both; a fibrinous exudation upon the pleura; in severe cases a more or less large collection of (serous, serosanguinolent, or purulent) fluid between the surfaces of the pleura, or between the gaps and in the meshes of the fibrinous exudation. In accord with the extent and location of the pleural effusion, there is more or less severe displacement of the contiguous structures.

I. DRY PLEURISY.

Stitch pain. Cough. Friction sound.

It is quite probable that many cases of dry pleurisy in young children escape detection. This is apt to occur especially in secondary pleurisy, where the symptoms of the original disease obscure those of the complication. Moreover little patients often refer the pathognomonic "stitch pain" to the abdomen instead of the side. Apart from the pain the subjective symptoms are few and mild. The child instinctively abstains from coughing and deep breathing, and, like an adult, lies on the affected side. As a rule, the diagnosis can readily be made on hearing the pleuritic friction sound—a dry, crackling sound on inspiration. The termination of dry pleurisy is either in rapid and uneventful recovery (sometimes leaving behind slight pleural thickening and adhesions) or in the graver form of the malady—*i.e.*, in pleurisy with effusion.

II. PLEURISY WITH EFFUSION.

A perceptible pleural effusion, be it composed of serum, blood and serum, pus or chyle may generally be recognized by the following distinctive features:—

Inspection.—Dyspnea with impairment of movement of the affected side.

Bulging.

Scoliosis.

In large effusions bulging of the affected area of the thoracic wall, and not rarely prominence of the hypochondrium of the corresponding side. Occasionally enlargement of the subcutaneous veins, and superficial edema. In cases of long standing in which the effusion undergoes partial or complete absorption, there is a lateral curvature of the spine with compensatory enlargement of the unaffected side of the chest.

Diminished vocal fremitus.

Palpation.—As compared with the healthy side, there is distention of the intercostal spaces on inspiration, and diminution of vocal fremitus. In large serous effusion fluctuation may be perceived by placing one finger of one hand in the intercostal space, and with the finger of the other hand imparting quick but gentle impulses to the fluid, in the direction of the other finger.

Diminished respiratory sounds.

Auscultation.—Varying with the amount of pleuritic effusion or thickening, the respiratory sounds may be diminished or absent over the affected side and exaggerated over the healthy portions of the lung. Where the effusion is small and the larger bronchi remain open for the respiratory current of air, we may hear distant bronchial breathing. In rare cases, especially in tuberculous pleuritic effusion, the respiratory murmur may simulate cavernous breathing and lead to errors in diagnosis, especially if the bronchophony over the compressed lung is transmitted along pleuritic adhesions or the chest wall.

Dullness up to flatness.

Percussion.—Dullness or flatness, corresponding to the amount of pleuritic thickening or effusion, over the affected portion of the lung, and often tympanitic resonance over the retracted lung tissue. Percussion must be performed lightly, for in the presence of only a thin layer of fluid forced percussion may elicit the normal resonance of the underlying lung. The sense of resistance to the finger is greatly increased. Displacement of the neighboring organs.

Grocco's sign (paravertebral triangle of dullness) is rarely elicited in young children.

With the establishment of the presence of a pleuritic effusion by means of the aforementioned physical signs, the nature of the pleural fluid content still remains to be determined. In the majority of instances this can readily be accomplished by means of exploratory puncture.

Except where the exudate is buried behind a thick pleural membrane or, more rarely, behind tumors of the chest wall (so that the needle does not reach the fluid), or where the pleural content is too thick to pass through the needle, exploratory puncture of a pleural effusion usually reveals any of the following fluids: Serum, serum with blood, serum with pus, pure pus, or chyle. In accordance with this finding it is customary to distinguish: Serous or serofibrinous pleurisy; hemorrhagic pleurisy; purulent pleurisy (empyema, pyothorax), and chylothorax.

Different fluids.

SEROUS OR SEROFIBRINOUS PLEURISY.

The onset may be sudden with vomiting, chills, rise of temperature and pain in the side, or, more frequently, insidious, —either as a primary disease with general malaise, short cough, increasing dyspnea and pallor, or as a secondary affection, with accentuation of the symptoms of the primary disease. In acute pleurisies the fever may be moderately high and persist for from two to three weeks, and then gradually subside, even though the effusion remains. Bilateral pleurisy is almost always tuberculous. Pleurisy associated with pericardial or peritoneal symptoms points to its tubercular character. In young children with a yielding thorax, absorption of large effusions is always associated with contraction of the affected half of the chest. The ribs become pressed together, the intercostal spaces narrow, the shoulder-blade is drawn nearer the vertebral column, and the latter twisted (scoliosis). With complete recovery from the disease, the deformity may in some cases gradually disappear. In the majority of instances, dullness and suppressed respiratory murmur continue as a result of pleuritic thickening.

Tuberculous.

Deformity of thorax.

The prognosis of this form of pleurisy except that due to tuberculosis is generally favorable. Occasionally acute pleurisy terminates fatally either as a result of a sudden excessive effusion or of pulmonary edema, embolism of the pulmonalis or of a cerebral vessel.

HEMORRHAGIC AND TUBERCULOUS PLEURISIES.

Irregular temperature.

Hemorrhagic effusion.

Tubercle bacillus.

Protracted cases of pleurisy should always be looked upon with suspicion. In very many instances they are of tuberculous nature. This is particularly true of bilateral pleurisy and of that with prolonged irregular temperature and a sero-hemorrhagic exudation. It is well to remember, however, that a hemorrhagic effusion is sometimes observed in scorbutic children, and that puncture of a blood-vessel or injury to the diaphragm or liver may bring forth blood in the aspirating syringe. In tuberculous pleurisy, before long, other symptoms of tuberculosis make their appearance. The presence of the tubercle bacillus in the exudate, or, if the lungs are involved, in the sputum, and positive tuberculin test settle the diagnosis.

PURULENT PLEURISY (EMPYEMA, PYOTHORAX).

Localization.

Owing to the frequency of pneumonias (the principal cause of pleuritic effusions) in children, empyema is of very common occurrence. In the majority of instances the exudation is purulent from the beginning, more rarely it is serous at first, and, after a protracted course, undergoes suppurative transformation, as a result of an endogenous infection by the pneumococcus, streptococcus, staphylococcus, or the tubercle bacillus. Pyothorax is usually unilateral, and localized on the left side more frequently than on the right. Occasionally it is bilateral, *e.g.*, in sepsis, pyemia, etc. Still more rarely it is multilocular, encysted, or interlobular. The amount of pus varies, from a few teaspoonfuls to a quart. The exudate may on the first puncture prove to be seropurulent, but as the disease advances the purulent character increases, becomes greenish-yellow in color, and sometimes fetid in odor. It may be feculent, indicating some connection with the abdominal contents.

Primary or secondary.

Pyothorax may develop primarily as a result of trauma. As a rule, however, it is met secondarily to inflammatory, especially suppurative, processes of the thoracic and abdominal organs, of joints, of ribs and vertebræ, or in association with general sepsis. As a sequel or complication of thoracic or abdominal diseases empyema usually sets in very insidiously, and may remain latent for some time until either the effusion is so large as to cause bulging of the affected side of the chest, or be discovered accidentally during a routine examination for some other ailment.

The onset is more acute in cases due to trauma, necrosis of neighboring bony structures, exanthematous diseases, or in sudden rupture into the pleural cavity of abscesses of neighboring organs (*e.g.*, hepatic, perinephritic, etc.). In such cases the symptoms resemble those of acute serofibrinous pleurisy, except that the temperature is higher and more irregular and emaciation and exhaustion are more pronounced.

Septic fever.

With early operative treatment empyema in children usually terminates in recovery. If let alone, the abscess may rupture spontaneously either in the lungs or externally through the chest wall—*empyema necessitatis*. The point of external rupture is usually found in the vicinity of the sternum, where the chest wall offers least resistance. If the rupture is in the lungs, a very large expectoration of pus occurs suddenly. In these cases there is always danger of pyopneumothorax. In another group of cases the pus may by inspissation lead to caseous residues and fatal issue from gradual exhaustion or from complications, such as tuberculosis, amyloid degeneration, etc.

Evacuation of pus.

Spontaneous rupture of abscess.

CHYLOUS PLEURITIS (CHYLOTHORAX).

Genuine chylous effusion in the thorax is an exceedingly rare condition. More frequently we meet with other milky effusions,—chyliform, latescent (non-chylous). True chylous effusion is the result of injury or obstruction of the thoracic duct, allowing the escape of chyle either directly through an opening in the wall of the duct or indirectly by transudation.

Chyliform.

The *differential diagnosis* between the different varieties of pleurisy can readily be made by means of exploratory puncture, and chemic, bacteriologic, and microscopic examination of the fluid obtained. Bilateral (usually tuberculous) pleurisy may be confounded with hydrothorax. The latter condition, however, is associated with anasarca, consecutive to heart or kidney disease, and generally runs an afebrile course. Left-sided pleurisy may be differentiated from pericarditis by the absence of heart-symptoms (triangular heart-dullness) in the former, and of lung-symptoms in the latter. The synchronous occurrence of both of these diseases, however, should be borne in mind. Right-sided, purulent pleurisy may be mistaken for an abscess or hydatid cyst of the liver. Careful examination will elicit the following differential points: In liver affections the midaxillary line forms the highest point of dullness, there is fluctuation, local

Differentiation from Hydrothorax;

Pericarditis with effusion;

Liver abscess;

Hydatid cyst,

tenderness and icterus; in pleurisy with effusion the last-named signs are absent and the midaxillary line forms the lowest point of dullness. Furthermore in pleurisy aspiration brings forth serum, blood or pus; in hydatid cyst of the liver, a non-albuminous fluid with "hooklets."

and Pneumonia.

The differentiation between lobar pneumonia and pleurisy is not always easy, since both diseases often coexist. In the latter event, however, exploratory puncture will readily clear up the diagnosis.

PNEUMONIA.	PLEURISY.
Dullness (late).	Flatness (early).
Temperature high.	Low.
Pulse-respiration ratio greatly disturbed.	Not so.
Bronchial breathing, bronchophony.	Suppressed breathing.
Vocal fremitus and resonance increased.	Diminished.

Dry diet.

Anodynes.

Treatment.—During the acute stage, keep the patient in bed. Limit the supply of fluids (in older children a semisolid diet, consisting principally of cereals, concentrated soups, beef-juice, soft-boiled eggs, etc.). Relieve pain by salicylates, perhaps, with some opiate internally; by strapping of the chest; flaxseed poultices, or the following ointment:—

℞ Tinct. iodini,
Olei gaultheriæ,
Olei terebinthinæ,
Guaiacolis,
Ichthyolis ....āā ʒss | 2
Liq. vaselini ....q. s. ℥j | 30

Sig.: Paint the affected parts twice a day, cover with absorbent cotton and bandage.

Aspiration.

Should the exudation increase to such an extent as to greatly interfere with breathing, aspirate and follow it up with the local application and strapping, and the administration of sodium iodid and infusion digitalis—the iodid to promote absorption of the fluid, the digitalis to counteract the interference with the heart's action by the exudate, as well as to stimulate diuresis. These latter procedures (except aspiration) are indicated also in cases running a protracted course, even without a large effusion. Aspiration should be practised in tuberculous pleurisy only to relieve the respiratory difficulty, and in chylothorax, both as a palliative as well as a curative measure.

As soon as pyothorax is detected, an immediate operation for removal of the pus is imperative. To wait for eventual spon-

Dangers of spontaneous evacuation of pus.

Operative treatment.

taneous evacuation of the pus through the lungs or externally is hazardous, principally because of the supervening, often fatal, exhaustion, and of the danger of complicating pyopneumothorax, an incurable fistula, or caseous degeneration. In tuberculous empyema, surgical interference is indicated only in threatening suffocation, or grave cardiac embarrassment. Empyema of brief duration with readily flowing pus usually does well with a free incision into one of the intercostal spaces and good drainage. On the other hand, cases of long standing or those with inspissated pus should be treated by resection of a rib, in order to permit free escape of the pus. The disfigurement after such operation in children is comparatively slight, and many cases of regeneration of even several ribs are on record. If the empyema is bilateral, it is advisable to operate at separate sittings.)

Fresh air.

Breathing exercises.

Patients recovering from pleurisy, with or without effusion, should have plenty of outdoor air, preferably in the country, seashore or mountains. Older children will derive great benefit from horseback riding. For expansion of the retracted lung after a protracted attack of pleurisy with effusion, systematic breathing exercises and cold sponging of the chest or cold affusions are very useful.

The importance of wholesome feeding should not be underestimated. Iron, the hypophosphites, cod-liver oil, and extract of malt are helpful to effect the cure.

Prompt attention to suppurative foci (*e.g.,* necrosis of ribs or vertebra) and early treatment of pneumonia by fresh air will frequently prevent empyema.

ASTHMA.

Stenosis of bronchial tubes.

The pathogenesis of asthma in children is essentially the same as that in adults,—stenosis of the lumen of the bronchial tubes. The stenosis may be brought about either by a spasmodic contraction of the muscle-fibers of the bronchioles, or by vasomotor turgescence and swelling of the bronchial mucosa. Children suffering from asthma usually present an hereditary tendency toward the disease, a susceptibility to protracted irritations of the nasopharyngeal, laryngeal, and bronchial mucous membranes, or a history of pertussis, bronchopneumonia or chronic bronchitis. In many instances local causes, such as adenoids, deformities of the nasopharynx, persistent thymus, etc., are met, and

Hay fever.

some cases are traceable to reflex causes, *e.g.*, indigestion. Symptomatic asthma is occasionally based upon *hay fever*—resulting from the action of pollen of certain grasses upon the mucous membrane of the nasopharynx—and, finally, an asthmatic attack is sometimes a manifestation of hysteria.

Night paroxysms.

With these etiologic factors in view, the subdivision of asthma into true and false is quite justified. Clinically the two varieties differ in that genuine asthma is invariably associated with chronic bronchial catarrh, hence, is based upon a pathological entity, and is of longer duration than false asthma. There is nothing characteristic about the catarrh. The paroxysm usually comes on at night. The child coughs, is a little wheezy, and in a few hours the typical attack is in full sway. The latter consists of extreme dyspnea, anxious expression of the face, congested eyes, cyanosis or pallor, cold extremities, restlessness and prostration. The patient is usually relieved by sitting up in bed. Auscultation of the chest reveals sonorous and sibilant râles, wheezing, squeaking, and whistling respiration. These sounds are often audible at a distance. As the attack subsides the breathing becomes less and less noisy, less labored, and less rapid.

Rapid, labored breathing.

Eosinophilia.

Recurrence.

Differentiation between true and false asthma.

There may be complete apyrexia, or a rise of temperature of from two to three degrees. The respiratory rate may be anywhere from 40 to 80 and the pulse 150 or over. During the height of the paroxysm there is marked eosinophilia, and where expectoration is abundant Curschman's spirals and Charcot-Leyden's crystals are found in the more or less glairy mucus. Toward the end of an attack the thorax may appear barrel-shaped; but unless the asthma is chronic in nature and characterized by prolonged attacks, the emphysematous deformity of the chest is usually only temporary. The attack may last minutes, hours, or days with temporary remissions, but after abatement of the paroxysm the child is apparently in good health except for the bronchial catarrh. In genuine asthma exacerbations usually occur in the fall and spring, when the sudden atmospheric changes contribute to catarrh of the mucous membrane of the respiratory tract. On the other hand, paroxysms of false, spasmodic asthma may occur at any time when the exciting cause, *e.g.*, indigestion, sudden fright, etc., presents itself.

As a rule, asthma is not fatal *per se.* Delicate infants, however, may succumb during a severe attack, as a result of suffocation, or after frequently repeated attacks, as a result of emphysema, cardiac dilatation, or even cerebral hemorrhage.

Attention to abnormalities of naso-pharynx.

The importance of curing the disease at its very inception or, at least, preventing or mitigating the paroxysm is obvious. A cure can be effected, if the cause can be found and corrected. Attention to abnormalities of the nose and throat is especially fruitful in this direction. Children having an asthmatic or arthritic history should be given particular care in the way of preventing colds and coughs, overfeeding, exposure to unhealthy surroundings, miasmatic affections, undue excitement, etc. An attack may, so to say, be aborted by early administration, preferably hypodermatically, of atropine $\frac{1}{2000}$ and morphine $\frac{1}{60}$ of a grain or occasionally by apomorphine gr. $\frac{1}{50}$ to $\frac{1}{100}$, repeated, if necessary, after a half an hour. The latter drug is especially efficient in "dyspeptic" or "hysterical" asthma. A few drops of a suprarenal gland solution instilled several times a day into the nose sometimes act admirably. If the paroxysm continues we may resort to the following combination:—

Morphine and atropine.

℞ Natrii iodidi	ʒss	2
Tr. hyosciami	ʒj	4
(Tr. quebracho	ʒj)	4
Ext. grindeliæ rub.	ʒss	2
Syr. pruni Virginianæ q. s. ad	℥ij	60

M. Sig.: ʒj every three hours for a child 5 years old.

Change of climate.

A course of syrup of the iodid of iron with cod-liver oil is very useful in all cases, and change of climate, to the seashore or inland, is sometimes effective in enhancing a permanent cure.

In treating asthma we should always bear in mind that asthma-like attacks are observed as a manifestation of a large thymus, malaria, or heart and kidney disease, calling for specific therapeutic measures to remedy the underlying affections.

Differential diagnosis.

ASTHMA.	SPASMUS GLOTTIDIS.	PULMONARY EDEMA.
Rare in infants.	Peculiar to infancy.	Moderately frequent.
Mostly of reflex origin.	Associated with rickets.	Secondary to cardiac debility.
Cough first dry, later loose.	Croupy.	Short and harassing.
Expectoration clumpy.	Not characteristic.	Frothy, bloody.
Difficult inspiration and expiration, whistling.	Inspiration, stridulous.	Inspiration and expiration. Subcrepitant, bubbling râles.
Duration, hours and days.	Minutes.	Minutes.

EMPHYSEMA PULMONUM.

Abnormal distention of the lungs with air occurs as a result of forced inspiration, *e.g.,* in stenosis of the larynx (croup) or bronchioles (asthma), whooping-cough, in bronchitis or bronchopneumonia with violent coughing, etc., or expiration, *e.g.,* cornet playing. Owing to the great elasticity of the puerile lung and its tendency to rapid adjustment, emphysema is rarely observed in children. If it does occur, it is most frequently limited to the apices and the anterior borders of the lungs. Exceptionally the emphysema is disseminated throughout the entire lung. In this event the symptoms are practically the same as those in the adult, to wit: Exaggerated resonance on percussion, dyspnea, barrel-shaped chest, and prolonged, incomplete expiration. In cases of long standing there is consecutive involvement of the heart—usually dilatation of the right heart, with or without hypertrophy.

Exaggerated resonance.

Barrel-shaped chest.

The treatment consists, in addition to removal of the cause, chiefly of change of air (mountains), light breathing exercises.

BRONCHIECTASIS.

Bronchial dilatation is not very uncommon in children, but as it usually forms a sequel of respiratory diseases (unresolved pneumonia) with violent coughing, or aspiration of foreign bodies into a bronchus, its presence is frequently obscured by the symptomatology of the preceding affection.

Cylindrical or sacculated.

The dilatation of the bronchus may be cylindrical or sacculated, and is almost always associated with peribronchial sclerosis (pulmonary contraction), and occasionally with emphysema.

Copious expectoration of two layers.

There are no pathognomonic signs of this affection except, perhaps, the copious morning expectoration of greenish-yellow, often fetid, purulent mucus, which on standing separates into an upper layer of serum and a lower of pus. Auscultation of the affected part of the chest reveals abundant moist râles, and if the bronchiectatic cavities lie near the chest wall, cavernous signs, which greatly resemble those of tuberculous cavities. In bronchiectasis, however, the sputum is free from tubercle bacilli and the course is usually afebrile and often remittent—the child often doing well for weeks.

Free from tubercle bacilli.

Relative recoveries from this affection are on record. The majority of cases are incurable, and after a shorter or longer (years) course the patients succumb to intercurrent diseases, such as pneumonia, miliary tuberculosis, or pulmonary gangrene.

The treatment, therefore, is principally hygienic and prophylactic: Wholesome food, tonics, breathing exercises, inhalation of warm vapors with eucalyptus, creosote or turpentine; residence in a high, dry region.

Creosote.

To facilitate emptying the dilated bronchi of their mucopurulent content, gentle inversion of the little patient a few times a day proves useful.

Inversion of patient.

PULMONARY GANGRENE.

Gangrene of the lungs is not rarely a sequel of pneumonia, phthisis, grave exanthematous diseases, gangrenous processes of the mucous membrane or skin, foreign bodies in the air-passages (entrance of bits of food), etc. The symptomatology of this affection is ill defined. In older children, as in adults, the macro- and micro-scopic appearances of the expectoration (upper layer, mucopurulent; middle, serous; lower, almost wholly of pus; remains of lung tissue and plugs containing needles of fat acids and detritus) are very helpful in the diagnosis. On the other hand, in infants chief reliance must be placed upon the general cachectic condition of the patient; the coexistence of gangrene of the mouth, throat or vulva; the frequent occurrence of hemoptysis (absence of tubercle bacilli), fetid diarrhea, and foul breath. The cough is usually spasmodic.

Fetid expectoration of three layers.

The course of the disease is comparatively rapid, fatal termination usually occurring within a few weeks, either from gradual loss of strength or from complications, such as hemoptysis, pneumothorax, thrombosis, or cerebral abscess.

Rapid course.

The treatment is symptomatic—tonics, inhalation of antiseptics, and if the gangrenous process is accessible, surgical intervention.

PNEUMOTHORAX, HEMOPNEUMOTHORAX, PYOPNEUMOTHORAX.

These conditions occur principally as a result of traumatism (fracture of a rib or clavicle), laceration of the lungs from violent coughing or by foreign bodies, perforation of the lungs through empyema, gangrene and similar destructive processes.

Traumatic origin.

The symptomatology is the same as in adults, thus: Sudden

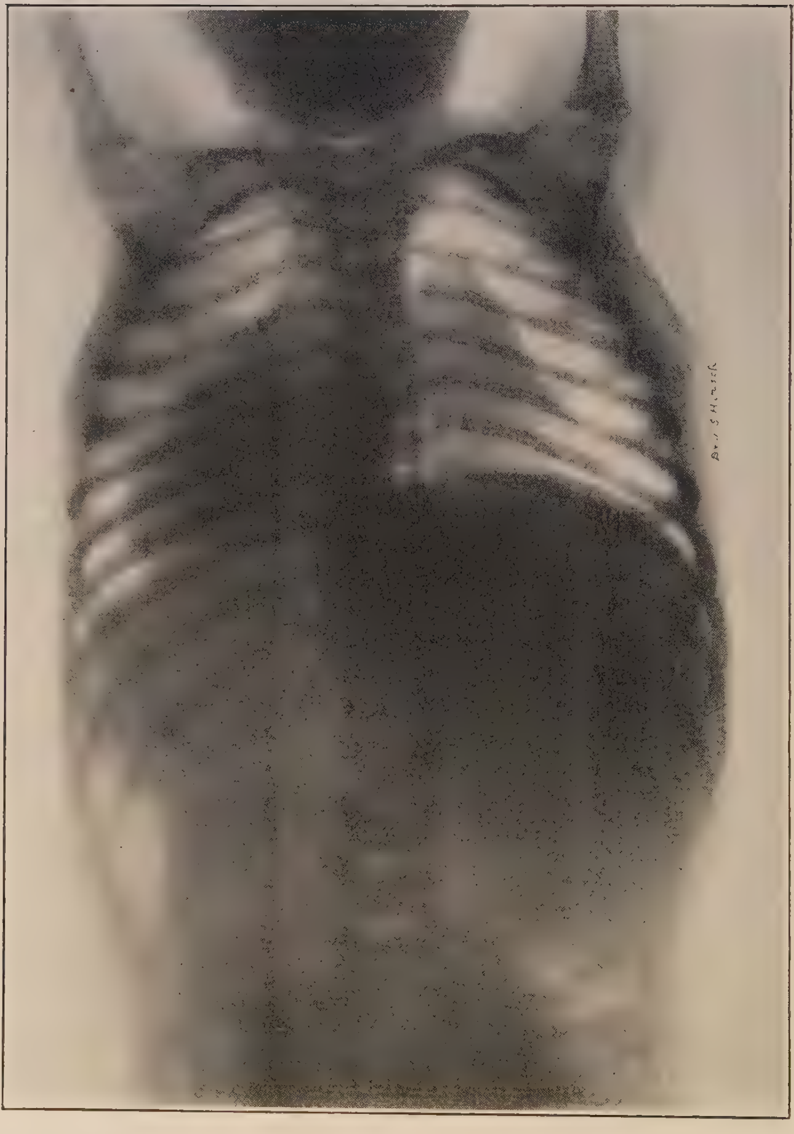

Fig. 74.—Pneumothorax. Note compression of lungs. (*Sheffield.*)

severe dyspnea, bulging of the affected side, tympanitic percussion sounds. When effusion occurs, there is hyperresonance over the upper portion of the affected part of the chest and dullness or flatness below the line of effusion. Succussion gives rise to splashing sounds. The diagnosis can readily be corroborated by thoracentesis.

Succussion sounds.

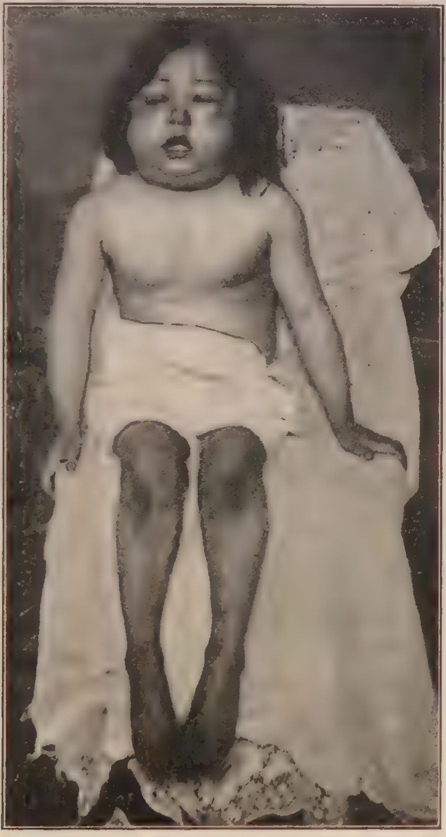

Fig. 75.—Pneumohypoderma[1] (five years old). The patient developed these symptoms suddenly during an attack of measles, with pneumonia. (*Sheffield.*)

Aspiration.

The treatment consists in the administration of opiates for the pain and aspiration (of air or fluid) to relieve the intense dyspnea.

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[1] See page 286.

PNEUMOHYPODERMA[1]

(Emphysema Cutis).

Result of violent coughing.

Entrance of air into the subcutaneous areolar tissue ordinarily results from rupture or laceration of pulmonary alveoli or bronchi during violent coughing or dyspnea (*e.g.*, in pertussis, measles, phthisis pulmonum), or secondarily to suppurative or caseous processes in the lungs. It is occasionally observed in connection with traumatic pneumothorax, and after tracheotomy and intubation. The air-inflation may remain limited to the neck or face or spread over the entire upper half of the body (see Fig. 75), and exceptionally also to the lower half.

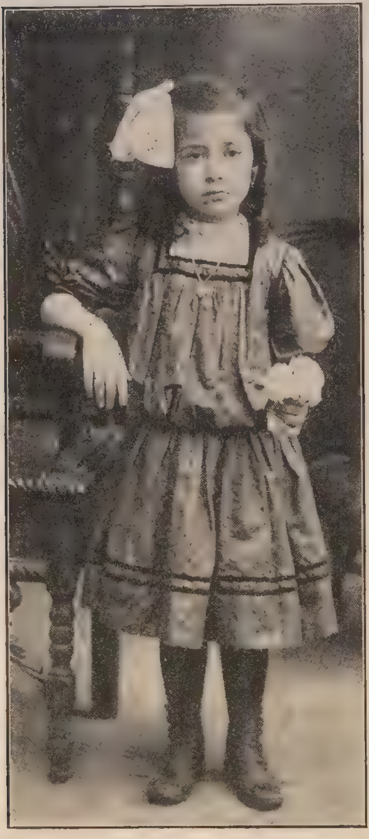

Fig. 76.—Same child as in Fig. 75, six weeks later. (*Sheffield.*)

Crackling perceived on palpation.

Pneumohypoderma can be detected by the distinct crackling sensation imparted to the examining finger, and can readily be differentiated from anasarca by the absence of pitting on pressure.

If the immediate cause can be promptly arrested, *e.g.*, violent cough by means of morphine, reabsorption of the air usually occurs within a few weeks. Rapidly fatal cases, however, are on record.

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[1] The new term is suggested because it indicates the exact seat of the trouble; it also helps to distinguish this condition from "surgical emphysema," which is produced by gasogenic bacteria.

CHAPTER IX.

Communicable Diseases.

INFLUENZA
(The Grip).

An epidemic infecti
Char. by cat. inf. of the
resp. tract, accom-
panied by a mucopurulen
discharge, fever, pain in
the muscles + prostrati

INFLUENZA is an acute, communicable, epidemic and sporadic disease due to the influenza bacillus of Pfeiffer and Canon. It is characterized by a variable group of respiratory, gastric or nervous symptoms, marked prostration and great tendency to complications and sequelæ.

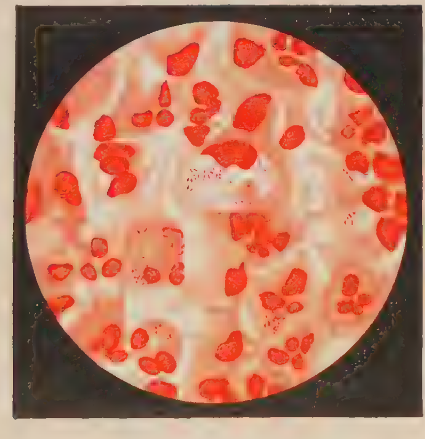

Fig. 77.—Influenza Bacilli. Sputum smear, stained with dilute Ziehl's solution. Bacilli chiefly intracellular, most of them show thickened ends. × 800. (*Lenhartz* and *Brooks*.)

Respiratory, gastric and nerve disturbances.

No age is exempt from this affection, and one attack neither predisposes nor immunizes against another one. The incubation period varies from two to five days; the onset, as a rule, is sudden, or may be preceded by a few mild prodromata common to all contagious and infectious diseases.

The attempt to classify the grip into three distinct types, to wit: catarrhal, gastric and nervous, is based upon an erroneous

Multiplicity of lesions.

Cough.

Sore throat. Prostration.

conception of the pathology and clinical data of this disease. On the contrary, it is the multiplicity of the lesions and symptoms which is characteristic of influenza. Thus, the child sneezes, coughs, has no appetite, vomits, complains of pain in the entire body, especially in the throat, head, and the lower extremities, is restless or lies exhausted in a semistupor for hours or days.

The cough is dry, loud, harsh, and painful (especially over the region of the sternum). The throat is deep red in color, and the tonsils and fauces are often covered with glairy mucus and occasionally with a yellowish-white, irregular deposit. In severe cases and especially in young children, large, soft or dry, sibilant râles are heard over a greater portion of the thorax, and, with the dyspnea and sometimes cyanosis, may readily be mistaken for bronchopneumonia.

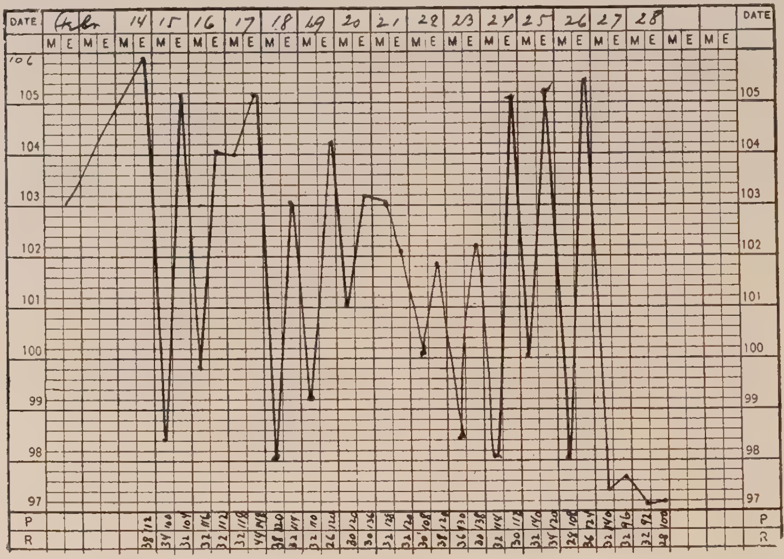

Fig. 78.—Fever Curve of Atypical Influenza in a child 14 months old. (*Sheffield.*)

Gastro-enteritis.

In infants particularly there are observed simultaneously more or less pronounced manifestations of gastroenteric involvement. The infant vomits, refuses breast or bottle, cries from pain of a colicky nature, and has an increased number of evacuations consisting of variously colored, undigested food. In older children the gastroenteric disturbance is much less marked—being limited to anorexia, nausea, vague abdominal pain, and sometimes constipation.

Paresthesia.

The nerve symptoms range from simple paresthesia, restlessness, dizziness and headache, to severe convulsions and profound stupor. In infants somnolence is more frequent than insomnia, and with the baby in a opisthotonos-like position (as a result of pain in the neck, trunk and extremities) one is frequently tempted to diagnosticate meningitis.

Influenza begins with abrupt rise of temperature of from 3° to 5° F., which runs an irregular course and ends by lysis or crisis, often accompanied by free perspiration, and intense prostration.

Atypical fever.

Occasionally the temperature with its concomitant symptoms may, without apparent cause, continue for weeks (see chart, page 78) and likewise suddenly cease. This type of the disease is often spoken of as chronic influenza, and is very apt to be mistaken for typhoid or malaria. Fortunately it is not commonly met in children. The majority of cases terminate within from three to eight days.

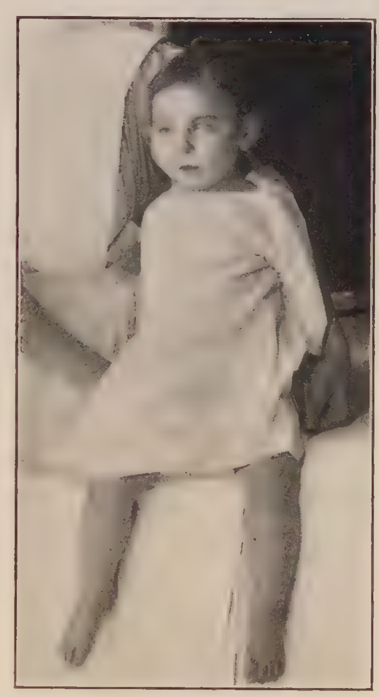

Fig. 79.—Paralysis of the N. Abducens, with convergent strabismus and slight facial paralysis, complicating an acute attack of influenza. (*Sheffield.*)

Delayed convalescence.

Convalescence is usually rapid in uncomplicated grip, especially in strong children and those free from hereditary or acquired encumbrances. In delicate and previously diseased children recovery may greatly be delayed by prolific complications and sequelæ. Pneumonias and otitides are especially common; and, frequently secondarily to these affections, and more rarely primarily, influenza may be complicated or followed by encephalitis,

Complications.

meningitis, paralysis, neuralgias, neuritis, nephritis and cardiac neuroses (*e.g.*, bradycardia).

Hemorrhagic processes.

The grip has a special predilection for hemorrhagic processes, such as hemorrhagic encephalitis, pleuritis or otitis, hemorrhages from the bowels, nose, skin, etc.—and occasionally gives rise to suppurative adenitis (especially of the submaxillary and parotid glands), rhinitis, conjunctivitis, periostitis, and more minor affections.

Every form of cutaneous eruptions may be met in this disease, and lead to erroneous diagnoses. In the presence of simple erythema, for instance, influenza may greatly resemble scarlatina and baffle the skill of even the keenest observer.

Influenza bacillus.

In doubtful cases of grip, especially in the absence of an epidemic, a correct diagnosis can be arrived at only by systematic scientific elimination of the suspected diseases and careful searching for the influenza bacillus in the expectoration or discharges from the nose, throat or ear.

Isolation of patient.

Tendency to nephritis.

Influenza is a treacherous disease and hence, however mild, the attack should not be neglected. Appreciating its high communicability and its tendency to many and grave complications, every effort should be made to arrest further spreading of the disease by strict isolation of the patient. Attention should be paid to the prevention of complications, principally pneumonia and otitis,—the first by avoiding exposure of the patient to bad atmospheric changes, the second by early treatment of the nasopharynx—which in the majority of instances serve as the causal factors of grip meningitis, and less serious complications and sequelæ. Daily examination of the urine is a highly commendable diagnostic and prophylactic procedure, especially in the so-called chronic grip which is prone to be followed by nephritis. Rest in bed should be enjoined as a means of prevention of cardiac disturbances.

Sodium benzoate.

The active treatment is chiefly symptomatic. The following combinations are quite efficient:—

℞ Natrii benzoatis	ʒj	4
Antipyrinæ	ʒss	2
(Codeinæ	gr. ss)	0.03
Syr. altheæ	ʒiv	15
Aquæ	q. s. ad ℥ij	60

M. Sig.: ʒj every three hours for a child 4 years old.

℞ Natrii benzoatis .... ʒss | 2
Aspirini .... gr. xv | 1
Olei sacch. menth. pip. .... q. s.
M. ft. pulv. no. viij.
Sig.: One powder every three hours for a child 6 years old.

℞ Antipyrinæ salicylatis .... ʒss | 2
Ft. pulv. no. viij.
Sig.: One powder every three hours for a child 6 years old.

Creosote.

Tonics.

For the acute cough ordinary mild expectorants will suffice. Protracted coughs usually yield promptly to creosote internally, and the tincture benzoin compound (ʒj to Oj of boiling water) by inhalation. Complications should be atended to at their earliest inception. Marked prostration calls for prompt stimulation by means of wholesome diet, small doses of strychnine, and digitalis. A sojourn in the country will materially aid in the prevention of dangerous sequelæ (*e.g.*, tuberculosis).

MEASLES
(Morbilli, Rubeola).

acute infectious disease, [illegible]
[illegible] eruption + [illegible]
[illegible] + conjunctiv
+ diffused
[illegible]
Isolated
(?) Bacillus
Canon + Piel

Measles is probably the most frequent and most readily communicable eruptive fever of childhood. Children of from two to six years old are most susceptible to it, but it is not rarely met in older and younger ones. In the majority of instances one attack immunizes the patient against another one; numerous exceptions, however, are on record. The cases of recurrent measles often prove to be rubeola on one occasion, and rubella, or a similar skin eruption, on another. The disease is communicable in all its stages by means of the contagium—which dwells in the lacrimal, nasal, and bronchial secretions, and probably also in the papules and squamæ—either by direct contact or, more rarely, through intermediate persons, the air or fomites.

Nasal catarrh.

Nine to eleven days—the period of incubation—pass after invasion of the system by the materia morbi without any characteristic manifestation of ill health, except slight anorexia, restlessness, ephemeral rise of temperature, etc., which toward the end lead to a more acute aggravation of the condition and mark the beginning of the prodromic stage. This stage usually lasts three days, rarely longer (up to a week in debilitated children). The little patient complains of chilliness, headache, and fatigue, hangs its head or sleeps most of the time, coughs and occasionally sneezes, and presents a rise of temperature of from 2° to 4° F. Not rarely the fever drops the next day, but the catarrhal symp-

Mottled palate.

Red spots on buccal mucous membrane.

toms continue in severer form. Examination of the mouth and throat in the majority of cases reveals upon the mucous membrane of the soft and hard palate diffuse redness or punctiform or stellate spots, and on the buccal mucous membrane from six to twenty, rarely more, red spots, with a central, rounded, slightly elevated, bluish efflorescence. These spots never cause pain or ulcerate. They are called Filatow or Koplik spots—the latter deserving the credit of having proven the pathognomonic significance of the spots as an early sign of measles.

Eruption.

Conjunctivitis.

Another twenty-four hours and the eruptive stage is reached. Bright red, pinhead- to lentil-sized dots appear over the forehead, about the ears and over the face (chin and around the nose and mouth—circumoral ring), and rapidly enlarge to irregularly serrated, pea- and bean-sized, sharply circumscribed, rounded or crescentic, slightly elevated red spots, which disappear on pressure. From these points the eruption rapidly spreads, often in crops, over the body and limbs, taking about twenty-four hours to complete the process. At this time the catarrhal symptoms also are at their height. The face is flushed; the eyes are red and watering and dreading light; the nasal catarrh is intense; the cough frequent, harsh and often barking; the voice hoarse; the temperature high (104° F., or higher); the urine scanty, high colored (diazo-reaction positive); the child is drowsy; at times delirious, often vomits and occasionally suffers from diarrhea (sometimes bloody). The peripheral and lymphatic glands are not rarely swollen and painful, and the spleen is somewhat enlarged.

Fine desquamation.

The eruptive stage lasts from five to six days. Toward the end of this stage the eruption begins to fade, especially on the face, and bran-like scales take the place of the exanthema. With the fading of the eruption there is often a critical decline of the temperature and concomitant symptoms, except the bronchial catarrh. The desquamative stage lasts about one week, so that the patient is usually entirely well by the end of the fourth week from the time of infection. Sometimes traces of the exanthema in the form of bluish-red spots remain over some portions of or the whole body which do not disappear on pressure with the finger. They are of no special significance.

Atypical forms.

Deviations from the typical course of the disease are not rare. Thus, the exanthema may be absent or so scanty as to escape observation—*morbilli sine exanthema*—notwithstanding the pronounced character of the catarrhal and febrile symptoms. In such cases the diagnosis from the grip is almost next to impossible, and can at best only be surmised in the presence of an epidemic or another case of measles in the immediate surroundings.

Resembles influenza.

The eruption may appear in the form of small papules, at times penetrated by a hair—*morbilli papulosi;* or be covered by minute vesicles—*morbilli miliares.*

Scarlatiniform.

Black measles.

The appearance of the exanthema may be delayed for a day or two and then be localized principally upon the body and limbs or become confluent so as to resemble the rash of scarlatina—*morbilli scarlatinosi.* Occasionally small hemorrhages occur between the spots—*morbilli hemorrhagici.* This form of measles is not to be mistaken for *morbilli hemorrhagici maligni,* "black measles," which is rather very rare and observed only in delicate, cachectic children. In this condition instead of the eruption there are numerous petechiæ and ecchymoses, in addition to hemorrhages from the nose, ears, genitalia, kidneys or bowels. (Malignant measles is usually associated with early depression, very high temperature, rapid and frequent pulse; dry, brown and thickly coated tongue; sopor, convulsions and coma, and often ends fatally within three days.)

Croup.

Pneumonia.

Occasionally the temperature is protracted or after a fall suddenly rises, indicating the occurrence or near advent of complications or sequelæ. (Ordinarily complications set in toward the end of the eruptive stage, but may appear as early as the prodromic stage. At this period also we are apt to find angina tonsillaris, epistaxis, severe vomiting and diarrhea, catarrhal laryngitis, pneumonia, etc.

Diphtheria.

Antitoxin.

In the eruptive stage pneumonia forms the chief complication.) Violent coughing is prone to give rise to laceration of the lungs and consecutive "pneumohypoderma" (see page 286). Quite frequently we meet also with pseudocroup and more rarely with diphtheria. The diphtheria of the throat sometimes develops secondarily to that of the conjunctiva; more frequently, however, the former occurs primarily, and the diphtheritic conjunctivitis remains limited to the original focus. It ~~was my~~ privilege to see two cases in point. One boy, six years old, succumbed to laryngeal diphtheria, while his brother, three years old, was saved from blindness and, perhaps, death, by early administration of antitoxin. The affected eye presented a clinical picture resem-

Noma.

Pertussis.

bling that of gonorrheal ophthalmia. The diphtheritic conjunctivitis cleared up entirely within ten days, but was followed by typical diphtheritic paralysis of the throat. Severe stomatitis is not uncommon, and numerous cases of noma (*q. v.*) complicating or following measles are on record. The same observation holds good for divers forms of ear affections. Measles is not infrequently associated with typhoid, erysipelas, varicella, scarlatina and acute pemphigus. The latter eruption may become gangrenous and prove fatal. The tendency to gangrene of apparently mild lesions of the mucous membranes and skin should always be borne in mind, as it is not at all rare to find general sepsis supervening just such lesions. Measles acts as a great predisposing cause to pertussis, which latter may prove fatal from rapid collapse or early supervention of bronchopneumonia. Sudden heart paralysis is rare.

Otitis.

Tuberculosis.

Among the sequelæ the following affections deserve special emphasis: Chronic conjunctivitis, keratitis, otitis, deafness, deaf-mutism, osteomyelitis, purulent pleurisy, or pericarditis, nephritis, tuberculosis, psychoses, meningitides and other nerve affections. Fortunately, most of the aforementioned complications and sequelæ are rare. Ordinarily measles runs a benign course. Still, measles should always be looked upon as a very serious disease, especially if it attacks very young and delicate children and those with a tainted hereditary disposition.

Isolation of patient.

The custom still prevailing with some ignorant people to congregate the children free from measles with those affected by it so that "they should all have it at once" is condemnable. Isolation of the patient should be insisted upon, and all other precautions available (see page 88) strictly adhered to.

Diaphoretics.

Anodynes.

The special measures in the treatment of measles consist principally of active diaphoresis by hot drinks, hot baths and diaphoretics (decoction of crocus, ʒj to Oss), and minute doses of an opiate and expectorants to relieve and loosen the cough. Attention to complications is all important, whether grave or mild. A light diet should be enforced as long as the temperature is above normal. The fear of free ventilation of the sick-room is unfounded. On the contrary, a liberal supply of fresh air should be allowed as a therapeutic measure. Where photophobia exists, the room should be darkened by shades.

The mouth and eyes should be kept clean with warm boric acid solutions, and the nasopharynx by instillations of a few drops of albolene.

Other symptoms arising should be treated according to indications.

℞ Liq. ammon. anisat.	ʒss	2
Spts. ætheris nitrosi,		
Syr. scillæ comp.,		
Tinct. opii camphoræ	āā ʒj	4
Syr. rhei	ʒiv	15
Aq. anisi	q. s. ad ℥ij	60

M. Sig.: ʒj every three hours for a child 4 years old. (Useful expectorant, etc.)

For differential diagnosis see page 327.

acute, contagious, eruptive
of short duration + mild
charac
(?) micro-orga

RÖTHELN

(German Measles, Rubella, Epidemic Roseola).

On superficial examination rötheln closely resembles measles, but on careful observation it is found to differ from it in so many respects as to justify its classification into a distinct disease. It is highly communicable and often occurs in epidemics. One attack is supposed to confer immunity for life; the exceptions to this rule, however, are by far more numerous in this disease than in measles. The incubation period lasts from ten to twenty-one days, and is generally free from any manifestations. There are none or very slight prodromata of from twenty-four to forty-eight hours' duration, consisting of languor, anorexia, and slight catarrhal symptoms. The eruption usually appears suddenly first on the face, and within from twelve to twenty-four hours over the entire body. Often it has disappeared from the face by the time the extremities are involved. The rash appears in *two* forms. One resembles that of measles—pale red papules, up to the size of a lentil, usually discrete, rarely confluent, and momentarily disappearing on pressure. The other form is finely punctuate and coalesces into diffuse rose-red patches—resembling the rash of scarlatina. The eruptive stage lasts from three to four days, and is usually free from severe general symptoms. During the height of the exanthema, there may be a rise of temperature of two or three degrees, but it is only of short duration. As in measles, the mucous membrane of the throat is the seat of diffuse or dotted redness; the buccal mucous membrane, however, shows no typical Koplik spots. Most patients complain of sore throat during the acme of the disease, but not nearly as much as in

Slight prodromata.

Morbilliform rash.

Scarlatiniform rash.

Angina.

scarlatina. The superficial glands, particularly those in the region of the angle of the jaw and less frequently those of the axilla, groin, etc., and the spleen are enlarged and tender.

Adenitis. Large spleen.

(The differential diagnosis between rubella and rubeola will be outlined on page 327. (Attention will here be directed, however, to the frequent, nay, almost constant, occurrence of free perspiration in rötheln, a symptom almost never met in genuine measles.) Where the rash is scarlatiniform, it may in the beginning be confounded with scarlet fever, but in the latter affection there are marked initial symptoms (vomiting!), high fever and pulse, and more severe throat manifestations.

Free perspiration pathognomonic.

Numerous so-called heat and stomach rashes greatly resemble German measles and it is not always very easy to tell them apart, particularly in the absence of an epidemic of rötheln. Under the circumstances it is safe to reserve the diagnosis for about twenty-four hours, and watch the results of a "cooling lotion" and a laxative.

For its differentiation from Duke's disease see page 327.

Rubella is considered the mildest of all acute exanthematous infectious diseases, and, as a rule, terminates favorably within one week from the onset of the symptoms. But in view of the occasional occurrence of serious complications (severe angina, bronchopneumonia, suppurative adenitis, and even meningitis), it should always receive proper attention, especially in the way of rest in bed, light diet, cleansing of the nasopharynx, and good hygiene. See also the treatment of measles, page 294.

Complications.

DIPHTHERIA

an acute inf. d. cau
by Klebs-Löffler, ba

Diphtheria is caused by a bacillus discovered by Klebs and Löffler in 1883. The bacilli are found in the secretions and excretions of the structures involved, and are transmitted usually through direct personal communication (kissing, etc.), but probably also through the agency of dishes, clothing, etc., and through a third person. The bacillus is very tenacious to life, so much so that rooms previously occupied by diphtheria patients and left vacant for weeks frequently harbor infective diphtheria bacilli, having resisted disinfection and prolonged ventilation.

Diphtheria bacillus.

The diphtheria bacilli have a predilection for the lining of the nasopharynx and larynx, especially of children of from two to eight years of age. By far more seldom they attack other parts of the body, *e.g.*, intestines, by extension of the primary inflammation. After imbedding themselves into the primarily affected structures the bacilli multiply and secrete their toxins, which enter the tissues and lymphatics and thence produce general infection.

Primarily local.

The incubation period varies from five to ten days. As a rule, the onset is sudden with vomiting, headache, chills, fever, sore throat, and difficulty in swallowing. Not rarely however it is preceded by indefinite signs of ill health of a few days' duration, consisting of anorexia, lassitude, slight fever, irritation of the respiratory tract, etc. In such cases the active stage of the disease may insidiously follow upon the prodromic stage without any pronounced variation in the clinical manifestations, the throat symptoms often remaining latent until discovered by a routine examination of the throat or unmasked by grave correlative symptoms. The importance of a routine examination of the throat of children in all kinds of complaints, therefore, is obvious.

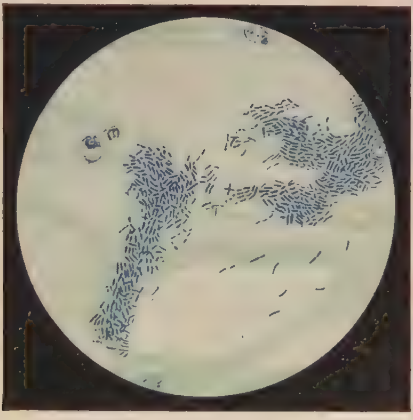

Fig. 80.—Diphtheria or Klebs-Löffler bacilli; smear preparation from tonsillar deposit. Löffler's stain. × 800. (*Lenhartz* and *Brooks.*)

The initial symptoms of the disease are not very characteristic, especially if the attack is mild. The uvula and tonsils are inflamed and somewhat enlarged. Careful inspection of the throat usually reveals upon the inner tonsillar or faucial surfaces a small, uneven, grayish-white, slightly elevated patch, or a few gray streaks or hemorrhagic specks. Within a few hours the deposit is found to have spread to the palatine arches and the

Deposit on inner tonsillar and faucial surfaces.

Raw, bleeding surface.

posterior pharyngeal wall, giving the appearance of a greenish-white, sharply defined, firmly adherent membrane, which if forcibly detached leaves behind a raw, bleeding surface, and re-forms very soon after. As the deposit assumes greater dimensions, the cervical and submaxillary glands, which at first are but slightly involved, become large and hard, assume the shape of large walnuts, and are very painful to the touch. Deglutition is difficult but not very painful—due to partial degeneration of the pharyngeal muscles and their nerves. The aforementioned constitutional symptoms continue.

Involvement of larynx.

Nasal sero-purulent discharge.

True croup.

The symptomatology thus far represents the first stage of a moderately severe attack of pharyngeal diphtheria. From now on three eventualities are possible: 1. The clinical picture may remain stationary. 2. The disease may spread to the nose. 3. The diphtheritic process may extend downward to the larynx. Since the introduction of the antitoxin treatment of diphtheria the number of cases falling into the first category has enormously increased. With early treatment the disease is rapidly arrested, the membranes are cast off spontaneously, and the patient makes an uneventful recovery within from four to eight days. Less frequently the second or third possibility occurs. Either as a result of extreme virulence of the infection or of negligence or improper treatment, the nose or larynx or both become invaded. In nasal diphtheria (*rhinitis fibrinosa et membranacea*), in addition to the previously mentioned symptoms, nasal breathing is obstructed and accelerated. The child keeps the mouth widely open, snores, is very restless, speaks through the nose, is almost unable to swallow, has fœtor ex ore, and coryza with a sero-purulent discharge. In laryngeal involvement (*diphtheritic croup*), symptoms of laryngeal stenosis predominate. The child's voice becomes husky, then hoarse, aphonic, and its breathing noisy, rough and wheezing, and as the disease advances it is attacked by a barking, croupy cough, dyspnea, retraction of the lower portion of the sternum and the ribs with each inspiration, and cyanosis. The dyspnea often occurs in paroxysms, which greatly resemble those of spasmodic croup (*q. v.*), and grow worse from time to time. Unless the air passages are promptly freed from the obstruction by intubation or tracheotomy, the patient passes into a state of stupor and finally succumbs to the effects of increase of carbonic acid and deficiency of oxygen in the lungs.

Both laryngeal and nasal diphtheria may develop primarily, and later become associated with pharyngeal diphtheria.

The course of the disease varies greatly with the location of the lesion, severity of the attack, and the period at which treatment is begun. Pharyngeal diphtheria usually pursues the most favorable course. Mild cases, as mentioned, may end in complete recovery in from four to eight days. In severer cases the symptoms may increase in intensity up to the fifth or sixth day, and then begin to abate, and after a rapid or protracted course finally subside. The same holds true of nasal or laryngeal diphtheria, provided treatment is instituted early and no complications supervene. Unfortunately in the latter form of the disease complications are of quite frequent occurrence. Exhausted from the prostrating effects of the paroxysmal attacks of laryngeal stenosis, the child is unable to withstand the onslaught of the diphtheritic poison (sometimes also mixed diphtheritic and streptococcic infection). The deposit, originally limited to the upper portions of the larynx, rapidly extends *downward,* involving the trachea and bronchi—leading to croupous bronchitis and pneumonia, and, as a rule, to a fatal issue—and *upward,* exerting its destructive action upon the pharyngeal, oral and nasal structures, often resulting in perforation of the palate, gangrenous sloughing of the uvula, etc. These cases of so-called *diphtheria gravissima s. maligna* sometimes develop very slowly and insidiously (*diphtheria larvata*) with symptoms of slight indisposition, slight rise of temperature, bronchial or gastrointestinal catarrh, and after a period of from a week to ten days are abruptly announced by true croup and the accompanying grave manifestations. Occasionally this form of the disease pursues a septic course right from the start,—irrespective of the location and extent of the deposit. It is characterized by vomiting, prostration, puffiness and earthy pallor of the face; small, often irregular pulse; epistaxis; bleeding from the mouth, pharynx or into the skin. The urine is scanty, loaded with albumin; the temperature may be slightly raised or below normal. Within from three to five days the child dies, in a state of low muttering delirium, from gradual exhaustion, or earlier from cardiac paralysis. On post-mortem examination, in addition to the diphtheritic lesions pathognomonic of all forms of the disease, the spleen is found enlarged; the kidneys, liver and heart in a state of cloudy swelling—a group of pathologic findings ordinarily met in severe infectious diseases—and,

Prognosis in pharyngeal diphtheria favorable.

Malignant form.

Latent form.

Septic symptoms.

Post-mortem findings.

varying with the intensity and number of complications, divers lesions in other organs of the body (*e.g.*, lungs, brain and alimentary canal).

There is nothing definite about the number and severity of the complications in any given case. As already stated, mild cases may become severe and exhibit all sorts of complications and sequelæ and, *vice versâ,* cases with severe onset may under proper treatment remain free from either and end favorably in a comparatively short space of time. Kidney, heart, lungs and nerve diseases form the most frequent complications and sequelæ. Transient albuminuria. Transient albuminuria is often observed even in mild cases. It usually begins the third or fourth day of the disease, sometimes earlier or later, and disappears with abatement of the other diphtheritic symptoms. Occasionally we find true *nephritis diphtheritica,* Nephritis. with large quantities of albumin and casts and more rarely also blood. The nephritis may also set in as a late sequel, during apparent convalescence, and remain more or less permanent. As a rule, however, the nephritis is of short duration, and rarely gives rise to local or general dropsy. By far more serious is the accompanying heart affection—so-called "heart Heart-paralysis. paralysis" from involvement of the pneumogastric nerve. It is often manifested by sudden heart-failure, and may set in either during the acme of the disease or any other time between then and as late as from four to six weeks after. It is apt to arise on the slightest exertion. The heart paralysis is not invariably sudden and fatal, however. Quite often it is preceded by heart-weakness with symptoms of dilatation—interstitial myocardial degeneration—such as extreme pallor; feeble, rapid, and irregular pulse; attacks of syncope, albuminuria, exhausting diarrhea, sometimes apathy, somnolence, sopor and death; or, less frequently, very slow convalescence, and gradual recovery, usually with remaining heart disease. Occasionally diphtheria is complicated Pericarditis. Endocarditis. by pericarditis or endocarditis. Bronchitis and pneumonia are especially prone to occur in laryngeal diphtheria, as a result of direct extension of the diphtheritic process to the trachea, bronchi, etc. (in intubated cases through entrance of foreign bodies, Aspiration pneumonia. particles of food, etc., into the air-passages—"aspiration pneumonia"), but also in other forms of the disease. The occurrence of pneumonia greatly mars the prognosis.

The most frequent sequel—occasionally also complication—of diphtheria is multiple neuritis, "diphtheritic paralysis." It is due

Multiple neuritis.

Regurgitation of fluids through the nose.

Strabismus.

Ataxia.

Hemiplegia.

to an intense degeneration of the peripheral nerves up to their roots. It follows in about one-tenth of all cases, probably mild and severe alike. It generally develops about the third or fourth week after the onset of the diphtheria, sometimes earlier or later, and affects the muscles of the soft palate by preference, causing a nasal tone of voice, and regurgitation of fluids through the nose. In combined esophageal and laryngeal paralysis there is also great difficulty in deglutition, not rarely giving rise to "aspiration pneumonia," as a result of entrance of part of the food into the air passages. These disturbances usually disappear spontaneously or on suitable treatment, within from four to six weeks. The paralysis may extend to the eye-muscles and cause strabismus, oculomotor paralysis, disturbance of accommodation and even total ophthalmoplegia. Less frequently the muscles of the trunk and extremities are implicated. The symptoms resulting are more or less identical with those observed in cases of multiple neuritis from other causes, and vary in intensity from simple motor weakness and ataxic gait up to hemiplegia. In severe cases the tendon reflexes and faradic irritability are entirely lost, and the muscles undergo atrophy. Nevertheless, recovery is the rule in the majority of instances, except when complicated by paralysis of the respiratory muscles (diaphragm) and the aforementioned baleful sudden heart-failure. As regards the hemiplegia, it is still uncertain, whether it is a genuine diphtheritic paralysis or caused by underlying alteration in the brain, such as cerebral hemorrhage, or cardiac thrombosis with embolism of the arteria fossæ Sylvii, since the hemiplegia not rarely begins with convulsions, loss of consciousness, and is often associated with aphasia and facial paralysis. If the patient survives the attack the hemiplegic symptoms usually subside within a few weeks, but weakness and contractures of the extremities may remain permanent.

Involvement of alimentary tract.

Less common complications and sequelæ are arthritides, otitides, pleuritis, peritonitis, suppurative adenitis, diphtheritic affections of the stomach, various rashes, etc.

From the foregoing discussion it can readily be appreciated that a positive prognosis is almost impossible. It should always be guarded, no matter how mild the case. The gravity of the epidemic, the severity of the attack, the strength and age of the patient, the quality of the heart, the period at which antitoxin has been administered—all have an important bearing upon the outcome of the case. However, no case should be despaired of, no matter how grave. Antitoxin treatment often performs miracles, even in apparently hopeless cases.

With the advent of the serum treatment, diphtheria has ceased to be the dread of the community. The mortality of diphtheria which previously ranged between 50 and 75 per cent., has now dropped to about 5 per cent. in pharyngeal and to 20 per cent. in laryngeal diphtheria—the earlier the serum treatment is begun with the lower the mortality. Indeed by administering diphtheria antitoxin at the very inception of the disease we are often enabled to limit the latter to its *local* manifestation—almost free from constitutional symptoms. Furthermore, those coming in close contact with the diphtheria patient may by means of from 500 to 1000 units of antitoxin be immunized against this affection for a period of from four to six weeks.

Immunization.

This procedure and isolation of the patient are the most potent prophylactic measures of diphtheria. As the nasopharynx forms the principal nidus for the development and spread of the diphtheria bacilli and their toxins, cleansing of the nasopharynx by means of mild antiseptics (instillation of Dobell's solution three or more times a day) will often aid in the prevention of infection. This prophylactic measure should be employed in conjunction with immunization by antitoxin, or without the latter—wherever there are contraindications to its use (*e.g.,* status lymphaticus, hemophilia) or objections on part of the family. Heart disturbances being the most dangerous complication of diphtheria, the heart should receive very careful attention, even in the mildest form of the affection. It should be examined daily, especially as regards acute dilatation of the heart. The patient should be kept under observation for at least four weeks after abatement of the acute course of the disease, and in the event of any untoward symptoms arising, immediately be put to bed and treated in accordance with the directions presently to be outlined. Even with an apparently normal heart it is imperative to keep the child perfectly at rest in bed for at least ten days after disappearance of the local symptoms. As to the prevention of "aspiration pneumonia," the reader is referred to the chapter on "Intubation."

Isolation.

Contra-indications to antitoxin.

Perfect rest.

The active treatment of diphtheria can be summarized in a few words: Counteract the diphtheria toxin; arrest the local lesion, and increase the power of resistance of the patient. When

Mode of administration.

Dose of antitoxin.

called upon to see a case of sore throat or laryngitis that is strongly suspicious of being diphtheritic in nature, we should immediately administer diphtheria antitoxin and lose no time in waiting for the results of a bacteriologic examination. The serum should be administered by deep hypodermic injections, a syringe somewhat larger than the ordinary hypodermic syringe being preferably employed for this purpose. The anterior surface of the abdomen or thorax or the outer surface of the thigh, where there is an abundance of subcutaneous cellular tissue, is generally chosen for the injections. Previous to the administration of the antitoxin the skin should be carefully washed with alcohol or some disinfecting solution and the syringe carefully sterilized. Nowadays the serum is obtainable in clean, hermetically sealed syringes, rendering their sterilization unnecessary. Children under two years of age should receive from 2 to 3000 units of antitoxin, and those over this age from 4 to 5000 units. Equal or smaller doses may be given after about eight hours, if no improvement is observed. In malignant cases,[1] or in those seen late, double doses should be administered at once and if necessary repeated. The effect of the serum is very beneficial, nay, sometimes magical. After a temporary rise, the fever often falls by crisis, the pulse improves, the membranes loosen and disappear, and the whole aspect of the case sometimes changes completely, for the better, within from eighteen to twenty-four hours. However, notwithstanding all that was said in favor of the antidiphtheritic serum, it is not always advisable to depend upon the serum alone.

Local treatment.

Resorcin-alcohol.

As diphtheria is originally a local affection and the secretion and absorption of the metabolic products (toxins) occur from the local lesion, the urgency of the immediate destruction of the bacilli at their point of entrance is self-evident. This is best accomplished by the different germicides and solvents, such as peroxid of hydrogen, strong solutions of carbolic or salicylic acid, 20 per cent. to 50 per cent. solutions of resorcin in alcohol, papain or pepsin, or the carbol-camphor solution referred to on page 242. Milder solutions of the same preparations should be used also for cleansing the nose—even in the absence of any lesion there. The local treatment should be repeated every two to four hours and continued until total disappearance of the acute symptoms of the diphtheria.

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[1] In desperate cases the antitoxin may be administered intravenously.

℞ Glycerit. papain.	ʒiv	15
Acid. carbolic.,		
Pulv. camphor.	āā gr. viij	0.5
Alcoholis	q. s. ad solv.	
Glycerini	q. s. ad f℥ij	60

This is applied to the throat by means of a cotton swab every two hours—changing the swab each time—diminishing the frequency of applications with the abatement of the severity of the symptoms.

Feeding.

Alcoholic stimulants.

The third indication, to increase the power of resistance of the patient, should be met by an abundance of nutritious, easily digestible food, stimulants and hematinics. Feeding of the little patient is as difficult as it is important. As a rule, total anorexia prevails, and it requires a great deal of patience and tact to induce the child to swallow a few mouthfuls of milk, broth, beef-juice, ice-cream, fruit-juices, etc. Still, much may be gained by administering the nourishment in small, frequently repeated quantities, and in small children, if need be, by rectal alimentation (peptonized milk). As a food and stimulant good wines and cognac are of inestimable value in diphtheria, especially in the septic variety. In malignant cases it should be given well diluted in large, frequently repeated doses (ʒj to ʒij every two hours) preferably by mouth, and in urgent cases in smaller doses also hypodermatically. It is advisable to employ mild stimulation from the earliest inception of the disease, and to continue it for weeks after in order to obviate—at least to a certain extent—sudden heart-failure. A useful combination which acts both as stimulant and hematinic, is the following:—

Strychnine.

℞ Strychninæ sulph.	gr. ¼	0.01
Liquor. ferri et ammon. acetatis	℥ij	60

M. Sig.: One teaspoonful every six hours, diluted in sweetened water.

Digitalis.

Whenever the local as well as systemic effect of iron is desirable, the iron and myrrh mixture referred to on page 319 answers the purpose admirably. Any untoward symptoms arising should be combated according to indications. In heart weakness strychnine and digitalis should be pushed to full tolerance.

In laryngeal diphtheria without nasopharyngeal lesions the local treatment outlined for the pharyngeal involvement may be dispensed with. Occasional cleansing of the nose and throat with Dobell's solution, however, is useful as a preventive measure.

It is of advantage also to have the patient inhale medicated vapors, such as the following:—

Inhalations.

℞ Thymolis	gr. x	0.6
Acidi carbolici	ʒss	2
Olei eucalypti	ʒj	4
Alcoholis	q. s. ad ℥ij	60

M. Sig.: ʒj in a pint of hot water.

Emetics.

Antispasmodics.

With early administration of antidiphtheritic serum the laryngeal stenosis rarely attains such severity as to demand relief by intubation or tracheotomy. Mild paroxysmal attacks of dyspnea often yield to emesis (ʒss of wine of ipecacuanha, or gr. 1/20 of apomorphine hydrochlorate), and a small dose of morphine (gr. 1/50) and atropine (gr. 1/500). But if these remedies fail, intubation or tracheotomy should be resorted to. It is always preferable to intubate (or tracheotomize) early than late. Whenever the dyspnea is steadily increasing in intensity and the temperature rises, this life-saving measure is indispensable, and procrastination is apt to prove fatal.

INTUBATION.[1]

O'Dwyer's invention.

For intubation as now performed the world is indebted to the late Joseph O'Dwyer, of New York. Intubation is employed in acute laryngostenosis whether of diphtheritic or other nature (see page 256). It consists in the introduction of a tube into the larynx, the size of the tube varying with the age of the child.

A set of intubation instruments (O'Dwyer's) suitable for children up to the age of puberty consists of six tubes, an introducer, an extractor, a mouth-gag, and a scale of sizes. O'Dwyer's latest tubes are made of hard rubber and lined with gold-plated metal. Each tube is supplied with an obturator, one end of which screws on the introducer. The tube is selected according to the age of the patient,—the smallest size for the first year, the second for the second year, the third for from two to four years, and the others, successively, for children two years older. It should be remembered that the tube must fit the larynx and the latter not be made to fit the tube.

Mode of Operating.—A tube of proper size for the child's age is selected, and, through the eyelet intended for the purpose, threaded with strong silk- or linen-thread,—long enough to reach the stomach and still protrude through the mouth. The thread is used as a precaution to prevent the tube from slipping into the stomach, in case it is wrongly placed into the esophagus instead of the larynx.

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[1] Partly after Graetzer and Sheffield's "Practical Pediatrics."

The obturator is then screwed tightly to the introducer and passed into the tube, and by repeatedly pushing the latter off from and replacing it upon the introducer we determine that the instrument is in working order.

Preparation of patient.

The patient is now placed upon a strong table and the body, from shoulder down, is wrapped snugly in a small sheet or blanket retained in position by several safety pins.

An assistant standing at the head of the table inserts the gag in the left angle of the child's mouth, well back between the teeth, and opens the gag as widely as possible without using too much force. The same assistant steadies the patient's head and holds the gag *in situ*.

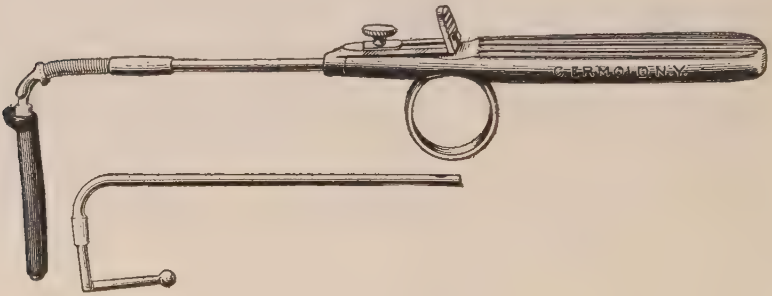

Fig. 81.—Introducer with Tube and Detached Obturator.

Introduction of tube.

The operator standing to the right and in front of the patient holds the introducer *lightly* between the thumb and fingers of the right hand, with the thumb resting just behind the button that serves to detach the tube, and the index finger in front of the trigger underneath.

The index finger of the left hand is now quickly passed into the pharynx down to the beginning of the esophagus and, by bringing the finger forward in the median line and raising and fixing the epiglottis, the tube is gently introduced, along the left index finger, into the larynx.

When the tube is inserted, it is slipped off by pressing forward the button on the upper surface of the handle with the thumb, while counterpressure is made with the index finger underneath. In removing the obturator the tube must be held down

Removal of obturator.

Removal of thread.

by placing the finger either on the side or posterior portion of the shoulder of the tube, lest the tube will be pulled along. After placing the tube in position the gag is removed, but the string is allowed to remain for about ten minutes, or until it is ascertained that the dyspnea is relieved and that no loose membrane is crowded down in the lower portion of the trachea. In removing the thread the finger is reinserted to hold the tube in place.

Localization of epiglottis.

If any difficulty is experienced in locating the epiglottis, it is better to seek the cavity of the larynx, a *cul-de-sac* into which the tip of the finger readily enters, and which cannot be mistaken for anything else. Once in this cavity, the epiglottis must be in front of the finger, and the latter is then raised and carried to the patient's right in order to leave room for the tube to pass beside it.

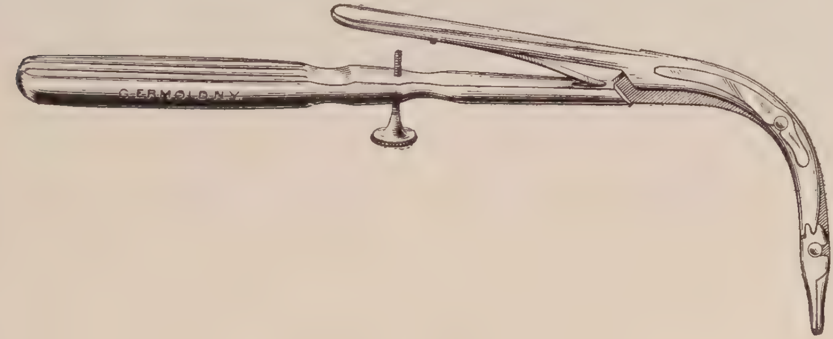

Fig. 82.—Extubator.

In the beginning of the operation the handle of the introducer is held close to the patient's chest, and then rapidly raised as the lower end of the tube passes behind the epiglottis; otherwise it slips over the epiglottis into the esophagus.

Feeding.

After-treatment.—The patient should be kept in a recumbent or upright position, but not allowed to lie upon the face or upon the nurse's shoulder, face downward. After about two hours feeding (in very small quantities) may be resumed,—nursing infants at the breast or bottle, and older children with semisolid substances, such as custards, matzoon, wine jelly, scrambled eggs, ice-cream, etc. It is of advantage to feed while the patient's head is lower than the body. The presence of the tube in the larynx does not contraindicate the use of emetics, which are sometimes needed when the bronchi are loaded with secretions.

Accidents and Dangers of Intubation.—With the experienced operator the principal danger that may attend intubation is asphyxia from existence of loose membrane below the tube, that is, in the lower portion of the trachea. If this occurs the tube should immediately be withdrawn and, after clearing the trachea of the membrane by induction of expulsive coughing or emesis, reinserted. There is rarely any danger from repeated failure to intubate, provided the operation is performed without such forcible manipulation as to produce a false passage, and the finger is not retained in the pharynx longer than ten seconds at a time, and the child is given a chance to get its breath between the attempts.

Loose membrane.

False passage.

Removal of the Tube.—The condition of the child being favorable, the tube is ordinarily removed after from five to seven days. This is accomplished with the patient in the same position as for insertion. The extractor is guided along beside the finger, which is first brought in contact with the head of the tube (be sure that the tube is still there!) and then carried to the right in order to uncover the aperture by lifting the epiglottis and to leave room for the instrument to enter beside it. Occasionally one succeeds in removing the tube by "stripping" the larynx from below upward with one hand and grasping the head of the tube between the index and middle fingers of the other hand.

Introduction of extractor.

Retained Intubation Tube (Prolonged Intubation).—Occasionally cases are met in which removal of the tube is immediately followed by asphyxia, though otherwise the patient seems in good condition. This is sometimes remedied by the use of sedatives internally and a spray of cocaine locally to relieve the spasmodic laryngeal stenosis, if present; or by gradual introduction of larger and larger intubation tubes anointed with some antiphlogistic drug (5 per cent. ichthyol).

Sedatives.

The Advantages of Intubation over Tracheotomy.—1. With an experienced operator, it is a bloodless operation. 2. No fresh wound is made which may prove a new avenue of infection. 3. No anesthetic is required, hence no shock or exhaustion from operation. 4. No skilled after-treatment is needed; no granulation wounds to treat.

Indications for tracheotomy.

Tracheotomy is indicated whenever the larynx is obstructed by foreign bodies, edema of the glottis, tumors (*e.g.*, multiple laryngeal papillomas, or compression tumors from neighboring structures) and cicatricial construction of the larynx. (See page 259.)

DIFFERENTIAL DIAGNOSIS.

1. Pharyngeal Diphtheria.—(*a*) *Pseudomembrane:* In pharyngeal diphtheria the pseudomembrane appears as a small, uneven, grayish-white, slightly elevated patch upon the *inner* tonsillar or faucial surfaces of the throat. The deposit—which contains diphtheria bacilli—augments by quick spreading, reaching within a few hours the posterior wall of the pharynx, and, in severe cases, the Eustachian tube, nares, and, more rarely, the conjunctiva. Anteriorly the pseudomembrane attacks the palatal arch and uvula. It may spread downward into the larynx or alimentary canal. The surrounding uncovered areas are grayish in color, due to overcrowding of leucocytes, nuclei, and mucus beneath. The tonsils, as a rule, are but slightly enlarged. The deposit, if removed, leaves a raw, bleeding surface and re-forms rapidly.

Extensive deposit.

In *follicular amygdalitis* the deposit begins as one or more white, small pellicles upon the middle or anterior portion of the tonsil. The pellicles, at first distinctly isolated, gradually coalesce, forming elevated patches. They are limited to the tonsils, may easily be removed, and reform slowly. The tonsil, usually one, is moderately enlarged, sometimes previous to the appearance of the deposit.

Small, isolated dots, gradually coalescing.

In *parenchymatous amygdalitis* the tonsil is greatly enlarged, often displacing the uvula. It is bluish in color and doughy in consistence. The deposit, at first white, soon becomes yellowish, resembling the "point" of an abscess.

"Point" of abscess.

In *herpetic amygdalitis* the deposit begins with minute vesicles, which have a tendency to burst and leave superficial ulcers. This form of amygdalitis is at times accompanied by stomatitis. Otherwise it resembles follicular amygdalitis.

Minute vesicles.

In *necrotic amygdalitis* the tonsils are moderately enlarged and the deposit lies deeply imbedded within the structure of the mucous membrane. The deposit, if removed, leaves behind a deep ulcer—sometimes gangrenous—surrounded by a distinct red zone; it spreads, as a rule, from one tonsil to the other by way of the anterior pillars and palatal arch, frequently attacking also the uvula.

Spreading ulcer.

(*b*) *Submaxillary Glands:* The submaxillary glands in diphtheria are greatly involved. They are large and hard, assuming the shape of a large walnut, and can easily be seen protruding from the angle of the jaw. They are very painful to the touch.

Large and hard.

Bilateral.

In *follicular and herpetic amygdalitis* the glands are moderately enlarged, softer in consistence and less painful to the touch than in diphtheria.

Moderate.

In *parenchymatous amygdalitis* the glands are moderately enlarged and diffuse, the swelling often extending as high as the ear.

Diffuse.

In *necrotic amygdalitis* the glands differ but slightly from those of diphtheria and cannot be relied on as a differential point of diagnosis.

Unilateral at first.

(*c*) *Early Constitutional Symptoms:* Except the presence of albumin in diphtheritic urine, none of the early constitutional symptoms are characteristic of diphtheria. Indeed, they are frequently less pronounced in diphtheria than in the other throat affections, unless the former is complicated by streptococcic infection. The temperature in diphtheria, as a rule, is moderate, about 101° to 103° F., and continuous. The pulse is feeble and quick and soon gives signs of exhaustion. The face, as a rule, is pale. Swallowing is difficult, but not very painful, due to partial degeneration of the muscles of deglutition and their nerve supply. Albuminuria is invariably present from the earliest beginning of the disease and is of great significance in the differential diagnosis.

Moderate temperature in diphtheria.

In the various forms of amygdalitis the temperature is quite high, especially toward evening, often reaching 105° F. The face is flushed. Deglutition is painful and difficult as a direct result of soreness and sensitiveness of the tonsils. Albuminuria is usually absent.

Hyperpyrexia in tonsillitis.

The diagnosis of *scarlatinal angina* is at best very difficult. It may be taken for granted that the primary amygdalitis is scarlatinal in nature, and that the sore throat setting in several days after is diphtheritic. It should be left, however, to the bacteriologic test to clear up the diagnosis.

2. **Laryngeal Diphtheria.**—Laryngeal diphtheria can only be mistaken for non-diphtheritic membranous laryngitis, also known as croup, which, on the other hand, is entirely distinct from spasmodic laryngitis (a mild catarrhal inflammation of the mucous membrane of the pharynx or larynx without the formation of a pseudomembrane). In speaking of non-diphtheritic membranous laryngitis I am fully conscious of the manifold denials made by advanced clinicians as to the existence of such a "non-diphtheritic" disease; here, again, I am

Differentiation from non-diphtheritic.

merely guided by the observations made in my own practice without attempting either to confirm or refute the views of others, and, while the exact distinction is associated with extraordinary difficulty, I believe to have been successful in making a correct diagnosis with the aid of the differential points referred to on page 255.

Differentiation from syphilis.

I may also mention that pharyngeal or laryngeal syphilis in childhood, if accompanied by an acute attack of amygdalitis, is apt to be mistaken for diphtheria. Early in the disease the history of the case, the usual presence of syphilitic manifestations on other portions of the body and the absence of diphtheria bacilli are reliable differential signs.

SCARLET FEVER

(Scarlatina, Febris Rubra).

Acute, contagious self-limited micro-org. not yet isolated.

Treacherous disease.

The more frequently one has occasion to observe and to treat scarlet fever, the more he appreciates the treacherous nature of this affection. Grave danger lurks often in the most benignly appearing attack, and dreadful surprises are not rarely encountered at a time when the patient is apparently at the threshold of recovery. It may be so mild in one child as to entirely escape observation, and yet may give rise to a most virulent type of the disease in another child. It is highly contagious and infectious in all its stages, the contagium (which is still unknown) being transmitted from person to person, through a third person, articles in use, toys, food and the air. Children of from 2 to 7 years are especially prone to contract the disease, but it has been observed even in newly born infants of mothers suffering from scarlatina just before delivery. As in other contagious and infectious diseases, some individuals possess an inherent or acquired permanent or temporary immunity against the disease. On the other hand, some children are highly susceptible to scarlatina and may have several attacks, sometimes even in the form of a relapse within from two to six weeks after the first attack (*scarlatina recurrens*).

Vomiting.

The incubation period of scarlet fever is ordinarily shorter than that of any of the other exanthematous febrile diseases. As a rule, it lasts only a few days (varies from one day to one or two weeks), and rarely gives rise to distinct symptoms of the approaching disease. On the contrary, often in the midst of apparently good health, the patient vomits (usually repeatedly), Sore throat.

Transient erythema.

Typical scarlet rash.

Circumoral ring, free.

"Strawberry" tongue.

complains of fatigue, slight sore throat, and chilliness, and young and nervous children are occasionally attacked by convulsions. The temperature rises up to 103° or 104° F., or higher; the pulse is greatly accelerated; the throat is deeply injected; the tonsils are somewhat enlarged and covered with a slight mucopurulent or hemorrhagic deposit. Sometimes a transient, prodromal erythema is observed on different portions of the body. The aforementioned symptoms continue for about twenty-four hours. By this time, or a few hours later, a bright-red rash becomes visible on the neck, chest and back and the flexor surfaces. On close examination the eruption is found to consist of very fine, rose-red to deep-red dots separated by minute, pale areas of healthy skin. The scarlet points are not elevated above the surface. The rash disappears on pressure, and when the finger-nail or a pencil is drawn across the reddened surface, a white line (*taches scarlatinale*) develops which remains *in situ* for a few seconds. This is due to increased contractility of the superficial arterioles. Gradually the eruption spreads over the entire body. It is least marked upon the face, and the circumoral ring —a space extending from the alæ nasi to the chin—is nearly always free from the exanthema. The affected skin is often edematous. With advent of the eruption the temperature rises, the submaxillary glands swell up, are hard and painful to the touch. Inspection of the throat in the majority of instances reveals a follicular deposit upon the tonsils which shows a tendency to coalesce and to form necrotic patches. The tongue is coated, very gray, and its edges and tip are bright red. The papillæ fungiformes soon project through the coating as red papules—"strawberry tongue." In accord with the height of the temperature, the patient is more or less thirsty, restless, delirious, refuses food, sometimes vomits; his urine is scanty, high colored, and usually contains a trace of albumin. The symptoms thus far related represent the clinical picture of typical scarlatina during the first two or three days of the *eruptive stage*. As the disease advances the gray deposit on the tongue is cast off, the entire tongue is more or less swollen, red, and covered with thickened papillæ. The deposit in the throat loses its tenacity, and often falls off *en masse*, leaving behind raw, sometimes bleeding surfaces. The pulse and temperature continue quite high (103° to 105° F.). Cases of considerable severity present in addition marked debility; febrile, cardiac, systolic murmurs; slight enlargement of the liver and spleen; at times somnolence, delirium, with or without high temperature. On the other hand, mild cases by this time may be on the road to recovery, free from fever and rash, ready to be around and about.

Desquamation.

The *stadium desquamativum* usually sets in four or five days after the appearance of the eruption, and depends somewhat upon the intensity of the exanthema, beginning earlier when the rash is pronounced. The peeling may vary from fine branny scales to large patches of epidermis, the coarser scales being usually limited to the hands and feet. Occasionally the nails shed with the epidermis. The peeling may last from two weeks to as many months, or even longer. In uncomplicated cases desquamation is followed by decline of the symptoms and convalescence.

Angina.

Complications are quite frequent, and their appearance is usually manifested by recrudescence of the temperature after defervescence. Scarlatinal angina—a necrotic inflammation of the throat—heads the list. It is caused by streptococcic infection and differs clinically from true diphtheria in that it almost never spreads to the larynx nor causes paralysis. Occasionally it is associated with true diphtheria.

Adenitis.

Gangrene of throat.

The throat involvement may be grave right from the beginning of the scarlatina, but more frequently it develops between the third and fourth days, usually in the form of an aggravation of the previous condition. The glands at the angles of the jaws swell enormously, are very hard and tender. Inspection of the throat reveals a large yellow or gray exudate on the greatly enlarged tonsils, and often also on the posterior pharyngeal wall. Scarlatinal angina often extends also to the nose, giving rise to a fetid, brownish-yellow discharge, and occasionally to deeper destructive processes and even to necrosis of the nasal bones. Scarlatinal angina is a very malignant affection, and frequently leads to a fatal termination as a result of gangrene of the throat, involvement of the neighboring large blood-vessels, purulent inflammation of the serous membranes (pleura, pericardium and meninges) extreme prostration, and general pyemia. In some epidemics one is able to distinguish two additional types of angina: 1. The "pestilential form," characterized by mucopurulent, foul masses in the throat and nose, spreading of the gangrenous process to the mouth and the mucous membrane of the lips and cheeks with consecutive hemorrhage, septicopyemic symptoms, increasing collapse, and fatal termination within about one week. 2. "Lentescent scarlatinal diphtheroid," which sets in about the sixth day of the disease with sudden rise of temperature, grave constitutional symptoms and intense swelling of the submaxillary glands. The local symptoms (which, by the way, are sometimes hidden!) in the nose and throat resemble those of true diphtheria, except that in scarlet fever there is a greater tendency to necrosis of the affected portions, and to perforation of the palate (as in syphilis). After stubborn persistence it quite frequently leads to fatal issue with symptoms of pyemia and asthenia. True diphtheria may be associated with any of the aforementioned forms of scarlatinal angina. An examination of the deposit for Klebs-Löffler bacillus, therefore, is always opportune. Purulent otitis frequently arises as an immediate sequel of the nasopharyngeal involvement by extension of the inflammation through the Eustachian tube and tympanic cavity. It is manifested by the usual symptoms of otitis media: earache, restlessness, rise of temperature, congestion and bulging of the drum membrane, and, as a rule, rapid perforation of the drum by the pus. In a great many cases the otitis leaves no serious consequences behind; in some of them, however, especially in those in which the escape of pus is delayed, scarlatinal otitis may lead to very grave consequences, such as deafness (in very young children deaf-mutism) mastoiditis, meningitis, etc.

Necrosis of palate.

Diphtheria.

Otitis.

Another serious sequel of the throat affection is *Angina Ludovici:* an inflammation of the submaxillary lymph-glands and the surrounding cellular tissue of the neck, extending from the submental region up to the mastoid process of the temporal bone. The inflammatory infiltration sometimes extends to the larynx and produces œdema glottidis, and, by gravitation, the pus may enter the mediastinum and neighboring structures (purulent pleurisy or pericarditis). It not rarely ends fatally with symptoms of septicemia, embolism or thrombosis.

Angina Ludovici.

Among the earlier complications of scarlatina we may mention also pneumonia, rheumatism (myositis, synovitis) and endocarditis. All of these complications are probably of septic origin. The pneumonia presents nothing characteristic, may be lobular or lobar in type. It usually runs a shorter course than primary pneumonia. Scarlatinal rheumatism occurs in two forms: Simple myositis, *i.e.,* a localized muscular infiltration, with sensitiveness on pressure, and vague "wandering" pain; and scarlatinal synovitis or arthritis which is manifested by pain, swelling and

Pneumonia.

Myositis.

redness of the joints, especially those of the fingers and toes; rise of temperature, and other constitutional symptoms. Sometimes several joints are affected by leaps. As a rule, scarlatinal rheumatism is benign in nature; occasionally, however, the joints may undergo suppuration, leading to general pyemia with fatal termination.

Suppurative arthritis.

Endocarditis.

In association with scarlatinal rheumatism, but often also without this, endocarditis forms a relatively frequent complication and sequel of scarlatina. Indeed the majority of cases of valvular heart disease in children, except, of course, those complicating primary rheumatic fever, are traceable to scarlatina. The endocarditis may at first be latent and escape detection, and again usher in with very grave symptoms, run the course of ulcerative endocarditis, giving rise to emboli and metastases in the liver, spleen, and kidneys, and end in sudden death or permanent valvular heart disease.

Nephritis.

The treacherous nature of scarlatina is most poignantly illustrated by the occurrence of nephritis as a complication. In the midst of apparently perfect health, at a time when the eruption has entirely subsided, either with or without any tangible cause (often after a slight error in the diet), the child is suddenly attacked by headache, dizziness, sometimes vomiting, and convulsions and examination of the urine reveals an interstitial inflammation of the kidneys. As the disease advances the symptoms enumerated under "nephritis" (*q. v.*) are rapidly and fully established. This complication usually occurs between the end of the second and third weeks. Hence the importance of daily examination of the urine in all cases of scarlatina, irrespective of the type or degree of severity of the disease. The duration of the nephritis varies greatly according to its severity, and the promptness with which it is discovered and treated. Ordinarily it lasts from two to four weeks and ends favorably, but relapses are not rare, and the nephritis may go on to chronic renal disease. In fact, scarlet fever, as a rule, forms the principal cause of chronic nephritis in children. Protracted scarlatinal nephritis often gives rise to hypertrophy of the left ventricle and occasionally also to dilatation of the heart with consecutive symptoms of ruptured compensation (recurrent anasarca, dyspnea, etc.). Genuine scarlatinal nephritis should not be confounded with the transient albuminuria not rarely observed during the first week of scarlatina, which most probably is due to the hyperpyrexia. As regards

Chronic Bright's disease.

uremia, and its grave accompaniments, the reader is referred to "Acute Nephritis."

More rare complications are the following: Stomatitis ulcerosa and aphthosa, noma, gangrene and diphtheria of the genitalia, orchitis, vaginitis, gangrene of the skin and of the tapering extremities; various nerve disorders, such as meningitis, hemiplegia, aphasia, tetany and psychoses; conjunctivitis, iritis, keratitis, choroiditis, neuroretinitis, retinitis albuminurica and sudden amaurosis.

Noma.

Aside from the sequelæ previously spoken of, scarlatina may be productive also of chronic purpura, chronic cutaneous affections (furunculosis), chorea, paralyses, marasmus and tuberculosis, etc.

For the differential diagnosis see table, page 327.

The discussion of the subject in question thus far relates principally to cases of scarlatina of ordinary severity. In these cases the diagnosis is usually quite easy, and the prognosis, except in the presence of serious complications, relatively favorable—provided, of course, energetic treatment is instituted early. We will now emphasize some of the numerous atypical forms.

Occasionally scarlatina is associated with an atypical eruption. Instead of the fine scarlet rash there may be variously sized papules or wheals upon a reddened base; minute vesicles (*scarlatina miliares*); or pemphigus-like blebs. The exanthema sometimes evolves gradually, requiring several days instead of hours as is the case in typical scarlatina. The rash may appear localized with intervening larger portions of normal skin (*scarlatina variegata*). Finally, there may be genuine scarlatina, with typical angina, nephritis, and even slight desquamation, without any exanthema (*scarlatina sine exanthema*). The diagnosis in all such cases is extremely difficult, and sometimes impossible, unless at the same time typical scarlatina prevails in the immediate surroundings, and the other symptoms point strongly toward this disease.

Atypical eruption.

Absence of exanthema.

The course of the attack also may present great variations. It may be so very mild and brief as to escape observation, or run a mild but protracted course, and remain free from complications. In the latter group of cases the temperature may be low, or remittent, with evening remissions and morning exacerbations (typus inversus). Fever may be entirely absent even in severe cases. Sometimes the temperature is very high (*hyperpyretic* *scarlatina*) from the beginning, giving rise to delirium, convulsions, etc., but subsides again after a few days, leaving the patient apparently unharmed. At other times very high temperature is characteristic of malignant scarlet fever.

Nerve-symptoms.

Scarlatina maligna, gravissima s. fulminans, fortunately is not of very frequent occurrence. In the majority of instances the grave manifestations are in full bloom within the first twenty-four hours of the onset of the attack. The child is suddenly seized with vomiting, rigors, delirium or convulsions; the temperature rises to 106° F. or even higher. The pulse is weak, rapid and irregular. Sudden collapse, coma, eclampsia and death follow in rapid succession (often within twenty-four hours). In another group of cases the course is more protracted, and typhoid in character. The temperature is not as high as in the aforementioned class, but is marked by evening exacerbations; the tongue is dry, the lips and teeth are covered with sordes, the abdomen is very tympanitic, and the stools are watery. The submaxillary glands are enormously enlarged. There are also signs of blood-dissolution, extensive hemorrhages from the nose, gums, and stomach, which greatly enhance the (fatal) exhaustion. The rash is usually of a violet color and hemorrhagic spots are scattered over the surface of the body. This form of scarlet fever is often spoken of as "septic, hemorrhagic scarlatina."

Malignant form.

Profound sepsis.

Hemorrhages.

Appreciating the unreliability of the initial manifestations, the uncertainty in the further symptomatology, the diversity of the course of scarlatina and its great tendency toward grave complications and sequelæ, it is prudent always to be very guarded in expressing an opinion as to the outcome of the disease, no matter how mild (or serious) the attack. The mortality varies in different epidemics, and depends partly upon the age (it is high in children under four and over ten years old) of the patient and principally upon the number and severity of the complications and sequelæ. In view of the high mortality it is essential to institute prompt prophylactic measures from the very inception of an attack of scarlatina. Rest in bed is indispensable even in the mildest cases, and should be enforced for at least two weeks (much longer in severe cases) from the beginning of illness. For about the same length of time should the diet be restricted, avoiding all such articles of food as are apt to upset the alimentary canal and to irritate the kidneys. In the active stage of the disease the diet should consist of milk only, and, as

Rest in bed.

Restricted diet.

the symptoms abate, light cereals and thin broths may be added; in older children also small quantities of toasted bread and butter, fish (boiled), chicken, soft-boiled eggs, and similar light food—all free from salt and spices. Easily digestible food should be continued for several weeks after subsidence of all traces of the disease. These procedures form the most potent means of prevention of renal and cardiac disease.

Attention to nasopharynx.

In view of the frequency of ear complications every effort should be made, firstly, by cleanliness of the nose and throat, to prevent infection of the Eustachian tubes, and secondly, infection arising, promptly to make a free outlet to the accumulated discharge (see Otitis).

Isolation of patient.

As regards isolation, room ventilation, and disinfection, see page 88.

It is quite difficult to formulate rules for the active treatment of the disease. Every case is a law to itself. We have no specific to combat the affection. Overdosing—but also underdosing—with medicines is to be deprecated. Very mild cases do best if left alone, except as regards prophylaxis.

Warm baths.

The average case being usually of medium severity, an attempt will here be made to outline a mode of treatment which is best suited to meet ordinary indications. The patient should be put to bed in a well-ventilated room (about 68° F.), the diet restricted to moderate quantities of water and a little milk—in the absence of vomiting. As at the onset of the attack vomiting is usually very marked, no medication per mouth should be prescribed, except, perhaps, a few minute doses of calomel and bicarbonate of soda. To relieve high temperature and nervous irritation, we order a warm bath every three hours. The baths have also a very salutary effect upon the kidneys by enhancing the elimination of the scarlatinal poison through the skin. Warm packs may be given instead of the baths. As soon as the vomiting has ceased, we increase the quantity of nourishment and direct our chief attention to the throat. The latter is swabbed every two hours with from 5 to 30 per cent. resorcin-alcohol solution or with the following:—

Local treatment.

℞ Acidi carbolici	ʒss	2
Pulv. camphoræ	gr. v	0.3
Alcoholis	ʒij	8
Glycerini q. s. ad	℥ij	60

M. Sig.: Apply to the diseased parts by means of a cotton swab every two hours.

The nose should be cleansed with Dobell's solution or similar antiseptic. If dysphagia and tonsillar swelling are marked, we prescribe moderate doses of sodium salicylate, or one of the newer salicylate preparations, and the following mixture:—

℞ Tinct. ferri chloridi,		
Tinct. myrrhæ	āā ʒss	2
(Kalii chloratis	ʒss)	2
Glycerini	q. s. ad ℥ij	60

M. Sig.: ʒj every three hours for a child 4 years old.

Diphtheria antitoxin.

Strychnine and digitalis.

Enteroclysis.

Morphine and atropine.

Lumbar puncture.

With the aforementioned therapeutic measures we are ordinarily successful to favorably proceed with the case up to the sixth day,—the time when "scarlatinal diphtheria" is prone to appear. As it is almost next to impossible to differentiate scarlatinal from diphtheritic angina, it is sound and safe practice to administer diphtheria antitoxin in all cases of severe angina, especially if an exacerbation of the symptoms occurs by the end of the first week of the disease. We usually inject 5000 units of antitoxin at once and repeat the dose as indications arise. The local and internal medicines should be continued, however, except bathing, which should be discontinued as soon as the temperature comes down to 100° F. The heart's action should be carefully watched, and any irregularity or debility detected, promptly treated by means of moderate doses of strychnine, digitalis or strophanthus. The latter two preparations are particularly useful in secondary involvement of the heart muscle. With the dietary and hygienic precautions taken, one is seldom confronted by grave scarlatinal nephritis. Ordinarily the symptoms are limited to slight albuminuria with occasional casts and blood-cells, which readily disappear upon the administration of a few doses of calomel and alkaline diuretics and diaphoretics, high flushing of the bowels and a few hot baths. But, as already suggested, occasionally the manifestations are extremely violent (delirium, convulsions, coma, etc.), resisting all sorts of medication, and growing worse from hour to hour. In these uremic conditions two therapeutic measures have proved to us of particular benefit: 1. Morphine and atropine hypodermatically. 2. Lumbar puncture. For a child four years old we may administer gr. $1/20$ of morphine and $1/500$ of atropine, to be repeated once or twice within twenty-four hours. In very bad cases both of these measures may be employed simultaneously. Their effect is often magical.

Where the uremic symptoms are slight, bromids with or without chloral per mouth or preferably per rectum suffice to relieve the nervous symptoms. As to the management of protracted cases of nephritis, see "Nephritis."

Bromids and chloral.

Simple transient scarlatinal myositis calls for no specific medication. On the other hand, arthritis demands prompt attention, since in the majority of instances it is a manifestation of sepsis and if left alone is apt to lead to general pyemia. The salicylates internally and ichthyol externally seem to influence it very favorably, and where these measures fail and pus forms we should resort to a free incision and drainage—but not too hastily. The same holds true for cervical adenitis which, though assuming very large dimensions, does not always suppurate.

Salicylates.

For suggestions as to the treatment of the remaining, less common complications of scarlatina, the reader is referred to the discussion of the respective diseases.

An extremely difficult problem confronts the attending physician when called upon to treat a case of malignant scarlet fever. Do what you will, the treatment is seldom of any avail. Early administration of antistreptococcic and antidiphtheritic serums sometimes saves life, and should always be employed, regardless of bacteriologic findings in the nasopharyngeal discharges. The same holds good for lumbar puncture, if meningeal symptoms predominate. High temperatures failing to yield to hot baths should be reduced by cold (68° to 70° F.) packs or baths. The heart should be kept actively stimulated by strychnine, strophanthus, digitalis, and suprarenal extract, the latter especially in hemorrhagic complications.

Antistreptococcic and antidiphtheritic sera.

During convalescence particular attention should be paid to the alimentary tract and skin. The bowels should be looked after, and stuffing the child with sweets, heavy meats, and alcoholic "tonics" strictly forbidden. The patient should be warmly clad and wear flannel or silk next to the skin. Exposure to sudden atmospheric changes should be avoided.

No alcohols.

To facilitate desquamation, the child should be given a hot soap bath every two or three days followed by oil inunction to prevent free distribution of the scales. The following combination is quite serviceable, and may be employed also in the eruptive stage of the disease to relieve itching and burning of the skin:—

Relieve itching.

℞ Thymolis	gr. x	0.6
Acidi carbolici	gtt. x	0.6
Alcoholis	ʒij	2
Liq. vaselini	℥ij	60

M. Sig.: For external use, p. r. n.

When desquamation is completed and there is otherwise no contraindication, the patient may be allowed out of doors. Cod-liver oil with the syrup of the iodid of iron and a sojourn at the seaside prove very helpful to rapid recovery.

Duration of contagion.

The patient is "contagious" for at least six weeks from the onset of the disease, hence, should not be permitted to mix with other children for that length of time or longer, if desquamation continues, or discharges from the nose, throat, vagina, etc., are present.

THE FOURTH DISEASE[1]

(Duke's Disease).

Slight efflorescence.

Mild course.

The existence of this affection is still awaiting authoritative confirmation. Some authorities maintain that it is merely a mild form of measles or scarlet fever. It begins after an incubation period of from 6 to 14 days with very mild febrile symptoms and an efflorescence on the face, including the circumoral ring. The next day the rash spreads, grouped in a sort of lacework arrangement, to the extremities and trunk. The course of the affection is conspicuous by absence of any severe symptoms and usually terminates favorably in from 5 to 8 days, without any specific medication.

Acute contagious disease of childhood charact. by an eruption [illegible] vesicles, [illegible] appear in successive crops [illegible] dif. parts of body.

VARICELLA

(Chicken-pox).

Nothing in common with small-pox.

The identity of the causal micro-organism of varicella is still unknown. It is absolutely proven, however, that it has nothing in common with the infectious agent of small-pox; hence an attack of chicken-pox confers no immunity against the former affection. The disease is communicable from person to person, through an intermediate person, through fomites, and the air. Children of from two to ten years of age are especially prone to contract the disease, but it is not rarely observed also in very young infants, and in children over ten, and even adults are not entirely exempt from it.

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[1] Termed so, being additional to the three known diseases: Scarlatina, Rubella and Rubeola.

Large vesicular eruption.

Several new crops.

Gangrene of skin.

Complications.

The incubation period lasts about two weeks, the last few days showing slight prodromata. Occasionally the symptoms of invasion are moderately severe. There may be vomiting, angina, conjunctivitis, transient ecthyma, considerable rise of temperature preceded by chill, and in small children convulsions. The eruption, which appears usually in small or large crops without any characteristic grouping simultaneously upon several portions of the entire body (also the mucous membrane of the mouth and throat), is fully established within twenty-four hours. At first the eruption appears in the form of slightly elevated rose-red spots, which disappear on stretching the skin. Within a few hours the center of the spot turns vesicular, filled with a clear fluid. The spots attain the size of a lentil or pea, but they may be larger, pemphigoid, and more rarely umbilicated. On the third day the vesicles usually collapse and desiccate, and become covered by brownish-black crusts. The latter usually fall off on the fifth or sixth day, leaving slight red spots which soon disappear. Repeated recurrences of new crops of the eruption in different stages of development (papules, vesicles and crusts), sometimes as late as ten to twelve days after the onset, are not rare and often serve of signal value in the differentiation of varicella from variola, in which latter disease the eruption remains uniform and stationary until the final stage of the disease. Occasionally the vesicular content is turbid or purulent (usually as a result of infection by scratching), and when the pustules heal leave behind scars resembling "small-pox pits." Sometimes the vesicles burst early and give rise to erosions and ulcerations which if occurring in the larynx may be productive of attacks of dyspnea and even fatal laryngospasm. The latter condition is of very rare occurrence. More frequently we meet, usually as a result of infection, with multiple ulcerative and gangrenous processes of the skin—*varicella gangrænosa*—in which the vesicles terminate in deep, foul-smelling ulcers, and extensive gangrene of the skin. This form of chicken-pox is most common in delicate, ill-nourished children and is apt to prove fatal. Complications and sequelæ in the form of nephritis—*nephritis varicellosa*—pneumonia, pleuritis pemphigus—*varicella bullosa*—multiple abscesses, pyemic processes (due to staphylococcic or streptococcic infection), icterus catarrhalis, dysentery, polioencephalitic manifestations, marasmus and even tuberculosis are on record, but they are rather of unusual occurrence.

[illegible]

As a rule, varicella pursues a benign and brief course, free from high temperature and any other constitutional symptoms, and rarely calls for any therapeutic measures. Rest in bed, careful diet, and local cooling lotions (2 per cent. thymol) or ointments (zinc oxid with 1 per cent. of salicylic acid and thymol) to relieve itching usually suffice in ordinary cases. Cleanliness of the mouth and throat. Attention to the urine. Finally, varicella is occasionally associated with other exanthema (*e.g.*, measles, scarlet fever). For additional differential points see table, page 327.

VARIOLA VERA. VARIOLOID

(Small-pox).

[illegible] are followed by a papular [illegible]

The history of small-pox is that of death and destruction. It is estimated that, before Jenner's discovery of prophylactic vaccination, one-tenth of all the children died of small-pox. On the other hand, with vaccination and revaccination rendered obligatory in most of the civilized countries, the occurrence of variola in a child is almost unheard of. If it ever does occur in successfully vaccinated children, the disease is usually mild, modified in form—*varioloid*.

Small-pox is an acute, highly contagious and infectious, endemic and epidemic disease, characterized principally by an eruption that passes through the stages of papule, vesicle, pustule and scab,—the development of the pustule being accompanied by a secondary fever.

Endemic and epidemic.

The nature of the small-pox producing poison is still unknown. It is undoubtedly a mirco-organism that exists in the eruption and probably also in the blood. The disease is most communicable during the pustular and desquamative stages—at which time mere entering the sick-room is said to infect one not protected by vaccination.

After an incubation period of from nine to fifteen days, which, as a rule, is free from any significant signs of illness, the patient is suddenly seized by a violent chill, fever, severe pain in the back, convulsions, delirium, prostration, and sometimes collapse and death—long before the appearance of the eruption. This mode of onset and termination is quite common in variola vera, affecting children under three years of age. Some cases survive until the appearance of a papular exanthema upon the buccal and pharyngeal mucous membranes, and then usually die from

Sudden, violent symptoms.

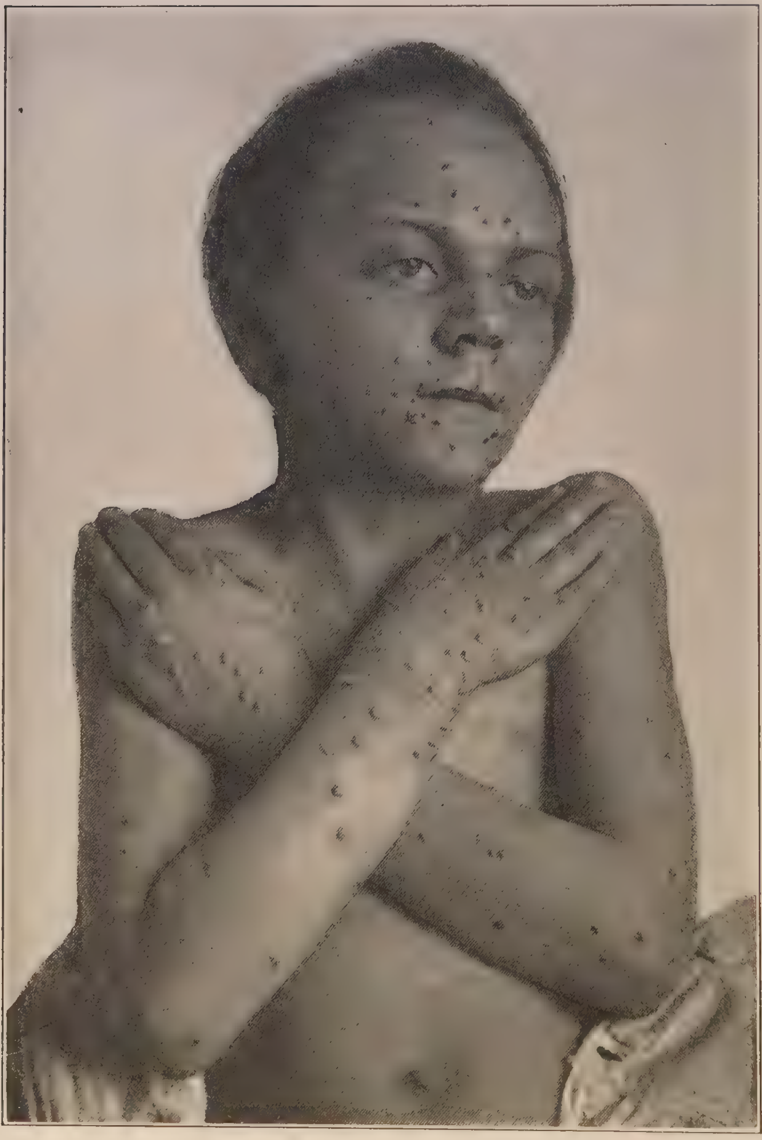

Fig. 83.—Mild Discrete Small-pox in an Unvaccinated Girl. Note absence of lesions upon the trunk. (Kindness of Dr. J. F. Schamberg.)

exhaustion; others again—usually older than three years—succumb to the attack in the suppurative stage, or, rather rarely, recover after a painful and tedious convalescence.

It is customary to distinguish three types of variola vera: Discrete, confluent, and malignant (hemorrhagic).

Discrete Form.—After the violent onset, the eruption, consisting of red, coarse spots, appears during the *third* day, first on the forehead and lips. The constitutional symptoms then abate, and the patient feels quite comfortable. On the *fifth* day of the disease the spots develop into papules; on the *sixth* day into vesicles which soon become umbilicated. On the *eighth* day the vesicles are transformed into pustules which emit a characteristic odor and on the *ninth* day they become entirely purulent and surrounded by a broad red band, the halo or areola, the face becoming swollen and the features distorted. On the *eleventh* day it is usually found that pus oozes from the pustules which on drying forms the scab or crust. The latter falls off some time between the seventeenth to twenty-first day, leaving a red, glistening depression or pit which soon changes into a white cicatrix. With maturity of the pustules (eighth or ninth day) the symptoms observed at the onset return—secondary fever. This fever of suppuration is the most critical period of the disease. In favorable cases the secondary fever abates after a few days and convalescence follows. The stage of suppuration is very prone to be complicated by severe inflammation of the larynx, bronchi,

Papules.

Vesicles.

Pustules.

Secondary fever.

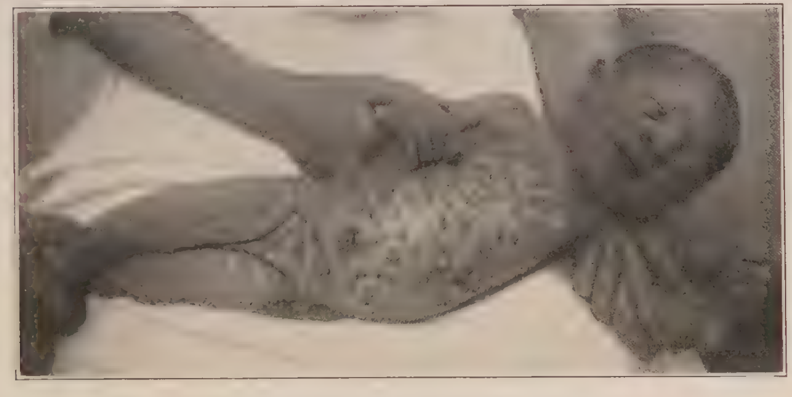

Fig. 84. -Fatal Small-pox in an Unvaccinated Four-week-old Infant. Seventh day of eruption. (Kindness of Dr. J. F. Schamberg.)

lungs, and serous membranes. As further complications or sequelæ we may mention stomatitis, noma, involvement of the eyes (phthisis bulbi), otitis media, dysentery and nephritis.

Violent constitutional symptoms.

Confluent Form.—It is characterized by extreme violence of the constitutional symptoms and by the confluence of the eruption at certain portions of the body, such as the thigh and lower portion of the abdomen (*Simon's triangle*) and the neck.

Black small-pox.

Malignant or Hemorrhagic Form.—This type of small-pox is characterized by malignancy and irregularity of the symptoms, and coexistence of hemorrhages and petechiæ. In this form are included the so-called black small-pox (*variola hemorrhagica pustulosa*) which usually leads to fatal issue in the suppurative stage, and the fulminant type of small-pox (*purpura variolosa*) which ends fatally within from three to four days.

Varioloid.

Mild, short course.

In contrast to variola vera with its dreadful consequences stands variola modificata or *varioloid.* The latter form of small-pox is usually observed in children rendered partially immune by previous vaccination or an attack of small-pox. Its course is shorter and milder than that of the other forms, the eruption is slight and devoid of suppuration,—hence its freedom from secondary fever and severe complications and sequelæ. The mortality in varioloid varies between 8 per cent. and 10 per cent. in infants and about 5 per cent. in older children.

Differential diagnosis.

Small-pox may be confounded, in the initial stage, with meningitis and, in the eruptive stage, with varicella and morbilli (especially morbilli hemorrhagici). Meningitis can readily be eliminated after a day or two. The differential signs between small-pox and the other exanthemata are outlined on next page. (See table.)

Vaccination.

Quarantine.

Stimulation.

If the patient with small-pox is seen early, vaccination should be performed at once; it may modify the attack. As a prophylactic measure it is also advisable to vaccinate all those who come and are apt to come in contact with the patient. Isolation, disinfection and preparation of the sick-chamber (the room should be kept dark by a deep-red shade) should be carefully carried out, in the manner described on page 88. The child should be confined to bed, and kept on a light but nutritious diet, and liberal supply of stimulants (wine, cognac). Especial attention should be paid to disinfection of the mouth and nasopharynx (mild solutions of potassium permanganate, or chlorate, peroxid of hydrogen). In high temperature and severe nervous phenomena pro-

TABLE OF THE DIFFERENTIAL POINTS OF DIAGNOSIS OF THE PRINCIPAL ERUPTIVE FEVERS.

	Rubeola	Rubella	Scarlatina	The Fourth or Duke's Disease	Varicella	Variola
Incubation Period (number of days).	9-11	10-21	1-10	6-14	12-14	9-15
Principal symptoms at onset.	Catarrh of nose and eyes; Filatov-Koplik spots.	Catarrh of nose and throat; spots on soft palate.	Vomiting, sore throat, hyperpyrexia; very rapid pulse.	Indisposition.	Slight febrile symptoms.	Headache, backache, chills, convulsions.
Time of appearance and character of eruption.	4th day; elevated red spots on face, spreading over entire body.	2d day; pale red maculæ on face and irregularly distributed over body.	2d day; bright red, pinpoint sized rash on neck, chest and face.	1st day; efflorescence on face; next day on extremities and trunk.	1st day; crops of thin papules, soon changing into vesicles which dry on 3d day.	3d day; coarse papules on forehead and lips, spreading downward, changing into umbilicated vesicles, pustules and scabs.
Conspicuous symptoms during course.	Moderate fever; tracheobronchitis.	Slight fever; glandular swellings; catarrh.	Hyperpyrexia, severe angina, strawberry tongue, desquamation.	None.	Pharyngeal and tracheal catarrh.	Disappearance of fever on 3d day; reappearance of "secondary fever" on 9th day.
Principal complications.	Pulmonary and ear disease.	The same.	Diphtheria; otitis; myositis; renal disease.	None.	Skin infections.	Respiratory disease; skin infections.

Warm baths.
Ichthyol.
Anodynes.

longed warm baths or cool packs act favorably. To prevent itching and extensive pitting we may apply 5 per cent. to 10 per cent. of ichthyol in equal parts of zinc and sulphur ointments, covered by some unctuous material to exclude the air. It is sometimes necessary to tie the patient's hands to prevent scratching; and to administer hypnotics and anodynes for the relief of restlessness and pain. The child should be quarantined for about six weeks.

℞ Antipyrinæ salicylatis	gr. xxiv	1.6
Tr. cinchonæ comp.	ʒiij	12.
Syr. aurantii	℥j	30.
Aq. aurantii ....q. s. ad	f℥iv	120.

M. Sig.: ʒss every six hours for a child 4 years old. (Antipyretic and anodyne.)

℞ Mentholis	gr. v	0.3
Bismuthi subgallatis	gr. x	0.6
Zinci stearatis	℥ij	60.

M. Sig.: Dusting powder to enhance desiccation of eruption and to relieve itching.

TYPHUS ABDOMINALIS

(Typhoid, Enteric Fever).

Frequent in children.
Atypical course.
Pyrogenetic stage.
Fastigium.

Typhoid fever is an endemic, epidemic, and sporadic infectious disease due to the bacillus typhosus of Eberth. It is characterized by a continuous, typical fever, gastrointestinal catarrh, and a roseolar eruption. It occurs probably as frequently in children (even fetal typhoid is on record!) as in adults, but owing to the mildness of the clinical picture it is frequently overlooked. The younger the child the greater the deviation of the fever from the usual course. Thus, the onset is either more protracted (with symptoms of subacute gastroenteritis) than in the adult or very sudden with chills and high fever. In older children the initial stage (pyrogenetic stage, first week) resembles that of adults and is marked by epistaxis, frontal headache, anorexia, furred tongue (later dry and brown), restless sleep, and gradual rise of temperature. The action of the bowels is not characteristic, and constipation may alternate with diarrhea (sometimes bloody). The fever reaches its height with the approach of the second week (fastigium), and varies in mild cases between 101° and 103° F. and in severe cases between 104° and 106° F., with morning remissions and evening exacerbations ("step curve"). Occasionally the typus inversus is observed, and not rarely the temperature is remarkably low throughout the entire course of the disease. The pulse is sometimes very frequent (160 to 180) but rarely dicrotic. The urine responds to the diazo-reaction, and contains traces of albumin. During this stage, the second week, the spleen is palpable, but not as distinctly as in adults. The roseolar eruption which usually appears about the eighth day on the abdomen, chest, back and limbs, is rather scanty and not rarely entirely absent. The typical eruption consists of small, elevated, rose-colored spots which momentarily disappear on pressure. They evolve in successive crops, each crop lasting about three days, and subside entirely after about ten days. Corresponding to the comparative mildness of the intestinal lesions, tympanites, iliac tenderness and gurgling are rarely marked. During the acme of the fever there are more or less marked nervous phenomena. Some patients are drowsy and apathetic; some are restless, shriek, and rave; some suffer from defective hearing, hyperesthesia, insomnia, or semi-stupor, and, finally, others may be dull during the height of the fever but otherwise be playful during the entire course of the disease. Children almost never present the status typhosus. As a rule, the blood gives a positive Widal reaction. (See page 102.)

Diazo-reaction.

Large spleen.

Rose-colored spots, in crops.

Apathy.

Widal test.

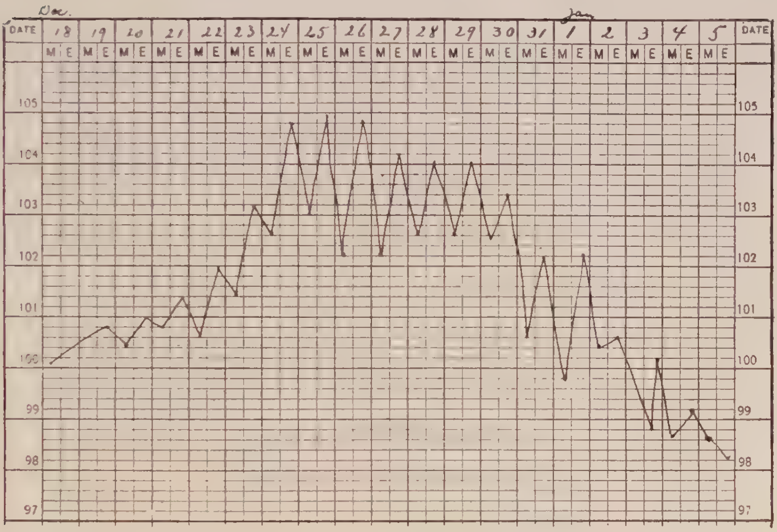

Fig. 85.—Fever Curve of Typhoid Fever in a child 4 years old. (*Sheffield.*)

With the beginning of the third week (defervescent stage) there is a decided improvement in the general symptoms. The

Defervescence.

tongue begins to clear at the edges, the appetite (often voracious) returns, the temperature declines, as a rule, by lysis, and the grave nervous symptoms gradually abate. (The temperature sometimes drops suddenly and remains normal or even subnormal.) In severe cases, however, the fever may continue (ambiguous stage) and with it all the other symptoms. Indeed, in older children the intestinal manifestations may become more pronounced, and hemorrhage from the bowels, perforation and peritonitis supervene. The usual bronchial catarrh may extend to the bronchioles and pulmonary tissue and lead to diffuse bronchopneumonia. Furthermore, improvement and recovery may be greatly delayed or entirely arrested by relapses, which are not uncommon between the third and fifth weeks, or by the following complications and sequelæ: Inflammation of the mucous membranes of the mouth (occasionally noma!), nasopharynx and larynx; parotitis, otitis, cutaneous abscesses, periostitis, perispondylitis (typhoid spine); pericarditis, endocarditis, purulent arthritis, pyemia, thrombosis and embolism; paralyses (usually neuritis), chorea, aphasia (lasts about a week), dementia, maniacal and melancholy states. The mental sequelæ usually consists merely of temporary irritability, hypersensitiveness, disposition to cry, capriciousness and surliness. On the other hand, cases of permanent mental aberration are on record. Typhoid fever is sometimes associated with pertussis, morbilli, scarlatina and diphtheria and in cases with a predisposition it is apt to be followed by pulmonary tuberculosis. Occasionally typhoid is followed by a post-typhoidal desquamation of the skin, and during and after an attack there is frequently a marked longitudinal growth of the bones, especially of the tubular bones of the lower extremities. As a result of it, the skin over these bones is transversely torn, the tears being indicated at first by red lines, and later by white scars.

Ambiguous stage.

Relapse.

Complications and sequelæ.

The aforementioned grave complications and sequelæ are very rarely observed in children. As a rule, the prognosis is favorable (less so in infants), and, even after severe attacks, convalescence comparatively rapid and uneventful. In young children the course of the disease is usually very brief, between twelve and fifteen days; in older ones it is nearly the same as in adults.

Mild intestinal lesions.

The morbid anatomical condition in the intestines is much milder than in adults; ulcers are rare, and, if present, are small, superficial and isolated; hence they heal without leaving behind any cicatrices in the intestines or any tendency to cicatricial contraction.

In view of these marked deviations of the clinical picture, the diagnosis of sporadic cases of typhoid often presents great difficulties. It is apt to be mistaken for simple gastroenteritis—febrile stage of shorter duration; spleen, in uncomplicated cases, not enlarged; diazo-reaction and Widal's blood test negative; influenza with pronounced intestinal symptoms—febrile "step" curve absent, nervous phenomena less pronounced, catarrhal symptoms more marked, Widal's test negative, the influenza bacillus in the expectoration; pneumonia—more sudden onset, more positive pulmonary physical signs, Widal's reaction negative, diplococcus pneumoniæ in the expectoration, neutrophilic leucocytosis; acute miliary tuberculosis—irregular temperature with sweats, hectic flush, often tuberculous sputum, more protracted course, Widal's reaction negative; tuberculous meningitis—lower temperature; slow, irregular pulse and respiration; trough-shaped abdomen; malaria—usually intermittent or recurrent fever, malarial plasmodium in the blood, influenced by quinine; septic endocarditis—pronounced heart symptoms, chills with septic temperature, absence of Widal's reaction. Occasionally typhoid begins with pain in the occiput, neck and back, opisthotonos, and other grave nervous phenomena, presenting the clinical picture of acute meningitis. The diagnosis in such cases is often almost impossible in the first few days of the disease. In doubtful cases the bacteriologic examination of the cerebrospinal fluid for the diplococcus intracellularis, and of the stools and urine for the bacillus typhosus often proves decisive.

Differentiation from: Gastroenteritis,

Influenza,

Pneumonia,

Miliary tuberculosis,

Tuberculous meningitis,

Malaria, and

Cerebrospinal meningitis.

As the contagium of typhoid fever resides principally in the gastrointestinal contents, it is imperative to thoroughly disinfect the stools and vomitus, as well as the linen and other articles in use that have been soiled by the discharges. Furthermore, by taking the precaution of boiling the drinking water or milk, excluding mosquitoes and flies from the sick-room, and by avoiding dissemination of the source of infection through soiled bath-tubs, hands, etc., the disease may be limited to a single patient notwithstanding the intercommunication between patient and other members of the family. Strict isolation, therefore, is not essential.

Disinfection of discharges.

Typhoid fever is a self-limited disease and not controllable by any specific measures. The treatment, therefore, should be

Hydrotherapy.

Opium in hemorrhage.

Liquid diet.

symptomatic, principally hygienic and dietetic. Cleanliness of the mouth and nasopharynx, cool sponging of the body, with water and alcohol or vinegar, or if the temperature is high, cool packs or full baths, at a temperature of from 80° to 90° F., and an ice-bag to the head, usually suffice to make the patient fairly comfortable. During the first few days we may administer small doses of calomel and bismuth, and later dilute hydrochloric acid, pineapple juice and some good wine or cognac. Hexamethylenamine is useful during the entire course of the disease. In intestinal hemorrhage, an ice coil to the abdomen and opium suppository (gr. 1/10 for every year of the child's age) will be found very efficient. Rest in bed should be enjoined for at least two weeks after defervescence. The diet should be fluid (milk with tea or a little cognac, soups, light gruel, chicken broth) during the acute course of the disease, and semisolid thereafter, care being taken not to overfeed. Transition to a more solid diet should be very gradual. Relapses call for the same mode of treatment as the original attack. During convalescence the different bitter tonics and iron are very desirable, and a sojourn at the seashore often works wonders.

Prevention of decubitus.

Complications should be carefully guarded against and immediately treated according to indications. Frequent change of position of the patient is usually effective to prevent serious pulmonary complications as well as decubitus. The skin should be hardened by alcohol, alum-water, etc., and as much as possible protected by air-cushions. The slightest abrasion of the skin should at once be treated by antiseptic dressings (2 per cent. solution of aluminum aceticotartrate).

Insomnia and excessive restlessness sometimes require hypnotics.

℞ Olei terebinthinæ	gtt. xvj	1
Olei menthæ pip.	gtt. iv	
Mist. acaciæ	q. s. ad ℥ij	60

Ft. emulsum.

Sig.: ʒj every four hours for a child 4 years old. (For tympanites.)

℞ Tr. nucis vomicæ	gtt. xvj	1
Acidi hydrochlor. dil.	ʒss	2
Aq. aurantii flor.	q. s. ad ℥ij	60

M. Sig.: ʒj, in water, three times a day for a child 4 years old. (Useful as a general tonic during the entire course of illness.)

ILEOCOLITIS EPIDEMICA
(Dysentery).

Inf. of large int., probably of [illegible] origin and
Charac. by pain, rect
tenesmus, & frequent pass
of mucus
blood -

This form of dysentery is entirely distinct from hemorrhagic enteritis or proctitis spoken of in connection with gastroenteritis on page 203. It is an infectious, epidemic and sometimes sporadic disease caused by the dysentery bacilli described by Shiga, Cruse and Flexner. The lesion is localized principally in the colon and less frequently in the ileum and rectum, and varies from a simple inflammation of the mucosa to a croupous, diphtheritic inflammation, with a membranous deposit, necrosis and ulcer formation.

Lesion.

In the majority of instances dysentery begins with simple diarrhea, without any constitutional symptoms and after from twenty-four to forty-eight hours is followed by the characteristic symptoms later to be spoken of. In some cases the onset is sudden with high fever and, in small children, with convulsions. Once the affection is established the symptomatology is quite pathognomonic: Colic, tenesmus, and bloody stools. The colic precedes and accompanies defecation and is followed by severe and prolonged tenesmus. The bowel movements vary between ten and thirty or more in twenty-four hours, and the dejecta consist either of pure blood or of blood and dirty, ragged shreds of tissue and fecal masses. (The abdomen is most frequently sunken, permitting palpation of the contracted colon.) The tongue is dry and heavily coated, the lips are cracked and covered with sordes, the appetite is lost, and the child suffers from intense thirst, and occasionally nausea and vomiting. As a rule, the temperature is raised (intermittent), but it may be normal or subnormal. After a few days the patient becomes greatly emaciated and prostrated, very anemic, and the expression of the face denotes great suffering. (Quite a number of children succumb during this stage of the disease; others again continue to battle for life and after a course of from seven to ten days begin to improve, the stools becoming less bloody and more feculent in character, the anorexia less marked, and the general condition much better.) (Relapses are not rare, and, when they occur, there is a great tendency toward the transition of the acute into a chronic process, with a very tedious convalescence, or death from exhaustion.

Colic, diarrhea, tenesmus and bloody stools.

Prostration.

Relapse.

Chronic form.

An attack of dysentery may be complicated by peritonitis,

Complications.

noma, parotitis suppurativa, abscess of the liver, fissura or prolapsus ani, pulmonary affections, etc., and may be followed by intestinal cicatrices and stenosis, paralysis of the sphincters, paresis of the extremities, and marasmus.

Amœbic dysentery.

Differentiation from enteritis with rectal involvement.

The very protracted cases of dysentery are usually found to be due to the amœba coli (entamœba dysenteriæ). The differentiation between this form of dysentery, that due to Shiga's bacillus, and catarrhal enteritis is important from the therapeutic point of view and can readily be made by a bacteriologic examination of the dejecta. Furthermore, it is well to remember that foreign bodies in the lower bowel may give rise to a group of symptoms similar to that of dysentery and that an inflamed prolapsed rectum, intussusception, an ulcerated rectal growth or hemorrhoids with coincident enteritis are very apt to mislead in the diagnosis. Careful examination (inspection and palpation) of the rectum disposes of these difficulties.

Disinfection of dejecta.

The patient suffering from dysentery, like one with typhoid, need not be strictly isolated. The dejecta and everything coming in contact with them, however, should be thoroughly disinfected. During an epidemic the drinking water, fruit and vegetables should be boiled, all modes of exposure to infection (mosquitoes, flies!) avoided.

Opium.

Acute dysentery calls for perfect rest in bed, an opiate (preferably hypodermically or per rectum) for the relief of pain, and light astringent diet (tea and toast, rice- and barley-soup or water). In the beginning the bowels should be cleansed with one moderate dose of castor-oil or syrupus rhei by mouth and one sterile cool water irrigation. The patient is then put on the following mixture:—

℞ Bismuthi subcarbonatis	ʒiv	15
Vini ipecacuanhæ	ʒj	4
Tinct. cinchonæ comp.	℥j	30
Mist. acaciæ	q. s. ad f℥iij	90

M. Sig.: One teaspoonful every two hours for a child 2 years old.

Irrigations with nitrate of silver; quinine.

In severe cases the intestines should be irrigated twice a day with 1:1000 of nitrate of silver, and once a day with 1:1000 quinine sulphate solution. The irrigation should be executed very gently by means of a soft-rubber catheter attached to an ordinary irrigator. Hydropathic applications to the abdomen (plain Priessnitz compress, or warm turpentine stupes) are useful.

Stimulation.

Collapse should be combated by local heat, cognac, red wine with a hot infusion of cinnamon, camphor, strychnine, etc. During convalescence care in dieting is still demanded, and the persistent anemia calls for iron, analeptics in the form of strengthening food (fresh eggs, milk with cereals, broths, etc.) and plenty of fresh air, and, whenever possible, a sojourn in the country, preferably at the seashore.

Complications and sequelæ require special treatment.

Tannates in chronic forms.

In chronic dysentery the tannates in conjunction with the quinine and silver irrigations do better than the bismuth preparations. Otherwise the management is the same as in acute dysentery. The more protracted the course, the greater the exhaustion and loss of blood; and the younger the child, the worse the prognosis. The mortality in different epidemics varied between 5 per cent. and 30 per cent. Early attention is a very great factor in reducing the mortality and the tendency toward chronicity.

MENINGITIS ACUTA

(Meningitis Cerebrospinalis).[1]

MENINGOCOCCIC, PNEUMOCOCCIC, TUBERCULOUS, STREPTOCOCCIC, ETC., MENINGITIS.

Primary.

Meningitis may be primary or secondary in nature. *Primary* meningitis may be the result of traumatism (may involve both the dura mater—pachymeningitis hemorrhagica—and pia mater, but usually the former) or be due to direct infection of the meninges by the diplococcus intracellularis meningitidis (Weichselbaum) and other pathogenic bacteria, *e.g.*, streptococci or staphylococci. *Secondary* meningitis is due to extension of the infection from neighboring or more remote parts. This form includes the tuberculous, or pneumococcus meningitis and the meningitides which are met with in divers acute infectious diseases, such as influenza, typhoid fever, erysipelas, otitis, diphtheria and the like. The infection spreads either by continuity (throat, nose or ear), by the lymphatics, or by the blood-vessels.

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[1] Our venturesque attempt to disrupt the time-worn, confusing mode of grouping of the different varieties of meningitis is based upon the following considerations: 1. The symptom-complex of fully established meningeal inflammation is practically identical in all forms of the disease, and differs only in the degree of mildness or severity of the attack, which depends upon the extent of the lesion, the susceptibility and the power of resistance of the patient to the microbic toxin and its baleful effects. 2. The same lack of distinction is observed in the pathological anatomy of the diverse forms of meningitis, except that in tuberculous meningitis we find local or general dissemination of tubercles in addition to the usual inflammatory process, which, however, are not manifested by special clinical symptoms. 3. Even the formerly accepted view as to the characteristic distribution of the inflammation in certain varieties of the affection, *e.g.*, the so-called "vertical" or "basilar" meningitis, etc., is no longer scientifically tenable in a strict sense of the word, since meningitis of the convexity of to-day may, by extension, be that of the base the day following and *vice versâ*. With these considerations in view, and appreciating also the fact that a positive differential diagnosis of the variety of meningitis can be made only by the findings of the etiologic factors in the cerebrospinal fluid obtained by lumbar puncture, we feel fully justified to discard the time-worn subdivision of meningitis into "serous," "purulent," "epidemic," "posterior basic," etc., and to classify the disease from an etiologic point of view. As we do of "tuberculous meningitis," we speak also of meningococcic, pneumococcic, streptococcic, influenzal meningitis, etc.—a classification which is not alone scientifically correct, but at once offers a clue as to the etiology, mode of treatment and prognosis.

Diplococcus meningitidis.

Secondary.

Meningitis is a disease peculiar to early childhood, the majority of cases occurring in the first three years of life. It prevails principally, often in epidemic form (cerebrospinal meningitis), during the late winter and spring months, at a time when, with rapid changes in the weather and crowding of the children in stuffy rooms, "colds" and their sequelæ are fiercely rampant. It is observed also sporadically during all seasons of the year. Delicate children are more prone to be attacked than robust ones, this being the case especially with tuberculous meningitis, which is frequently the culmination of latent tuberculosis of other organs of the body.

Epidemic.

Sporadic.

The mode of onset of the disease varies greatly. It is usually abrupt in primary meningitis, rarely preceded by a few indefinite signs of ill health, such as anorexia, restlessness and headache. In secondary meningitis the attack, as a rule, develops more insidiously and is often obscured by the symptomatology of the preceding affection. Meningitis supervening latent tuberculosis with few exceptions is particularly prone to be gradual in its development. In these cases the child may for weeks manifest apathy, anorexia, vomiting, wasting, occasional rise of temperature, and other symptoms corresponding to the seat of the original lesion (*e.g.*, caseation of the bronchial, mesenteric, or intestinal glands; bone or joint disease, etc.).

Slow onset in tuberculosis.

Acute meningitis, be it primary or secondary, gives rise to dizziness, headache, nausea, projectile and usually persistent vomiting, rise of temperature, jactitations up to convulsions, alternating with drowsiness, stiffness and pain in the neck. This group of symptoms while *per se* not at all characteristic is nevertheless strongly suspicious of the disease. Finding a patient in this condition we should at once carefully examine him for

Headache, projectile vomiting and convulsions.

the following more or less pathognomonic physical signs and symptoms of meningitis:—

Neck symptom.

Rigidity of the Neck.—This symptom is elicited by placing the hand under the patient's occiput and flexing the head upon the chest. In meningitis the neck will be found stiff and painful. Forcible flexion of the head upon the chest usually produces synchronous flexion of the legs upon the abdomen.[1] The child instinctively assumes a lateral position, as the dorsal position proves very painful by pressure of the pillow against the head. Rigidity of the neck is present at one time or another in all cases of meningitis. It is especially pronounced in cases in which the inflammation begins at the posterior part of the brain. As the disease advances the rigidity extends to the muscles of the back and extremities, giving rise to a spasmodic rigidity of the body in which the trunk is arched forward and the shoulders and buttocks are thrown backward while the legs as a rule are flexed upon the thighs—opisthotonos. Occasionally the forearms are extended and the fingers clinched in the palms.

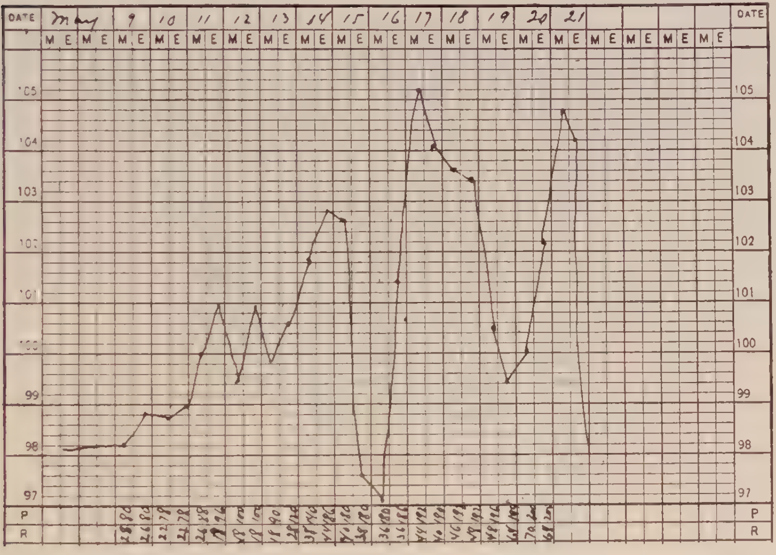

Fig. 86.—Fever Curve of Tuberculous Meningitis in a child 2 years old. (*Sheffield.*)

Opisthotonos.

Photophobia.

Changes in the Eyes.—Intolerance to light and contraction of the pupils form early symptoms of meningitis. Dilatation or inequality of the pupils is usually met with later. The inequality is usually transient and variable, present at one time and absent at others; now one pupil, now the other may be the larger. Strabismus and nystagmus are observed in advanced stages of the affection. Examination of the fundus reveals in the majority of cases of tuberculous meningitis optic neuritis or papillitis, and tubercules in the choroid. Optic neuritis is occasionally found also in other varieties of meningitis, chiefly when the base is involved. After the first week the child often keeps the eyes open staring immovably into distance.

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[1] Brudzinski's neck sign.

Optic neuritis.

Vasomotor and Cutaneous Disturbances.—Cutaneous irritation is usually followed by a vivid and enduring congestion of the skin—*Taches Cerebrale* (Trousseau's sign). This symptom is not very significant, being observed also in other infectious diseases, *e.g.*, typhoid fever. Eruptions of the skin—erythema, herpes, urticaria and purpura—are quite frequent. Purpuric spots are especially common in fulminant cases (hence often spoken of as spotted fever). They vary in seat and may coalesce to form dark diffuse extravasations into the skin.

Taches cerebrales.

Spotted fever.

Kernig's Sign.—This symptom consists of inability of the examiner to extend the patient's legs with the thighs flexed on the abdomen. It is met in the majority of cases of meningitis, but is not pathognomonic of the disease, since it is observed also in other affections, *e.g.*, typhoid fever, and occasionally also in normal infants. In conjunction, however, with the other meningeal symptoms Kernig's sign is very helpful in the diagnosis.

Kernig's sign.

Reflexes.—In the early stages of meningitis the skin and tendon reflexes are somewhat exaggerated, but with the gradual loss of muscular power they disappear partially or wholly.

Babinski's Reflex.—Irritation of the plantar surface of a patient suffering from meningitis produces extension of the great toe with flexion of the other toes. It is a characteristic sign of disease of the pyramidal and lateral tracts of the cord, hence is more apt to be observed in very diffuse forms of inflammation of the meninges and underlying structures (tuberculosis) than in the localized forms of the disease. This sign is least reliable in infants under two years of age, but is of corroborative value in older children.

Babinski's sign.

Leichtenstern's Symptom.—This consists of lightning-like contraction of the whole body on striking any part of the bony framework with the percussion hammer. It is a symptom of meningitis, principally during the stage of irritation.

McEwen's sign.

McEwen's Sign.—With the patient in an upright position and his head inclined to one side, percussion over the junction of the lower portions of the frontal and parietal bones gives a tympanitic note. This situation corresponds to the anterior horn of the ventricle, and the note is caused by the presence of fluid in the ventricle. Hence it is most frequently observed in the tuberculous variety of meningitis. This sign is not pathognomonic before complete ossification of the skull.

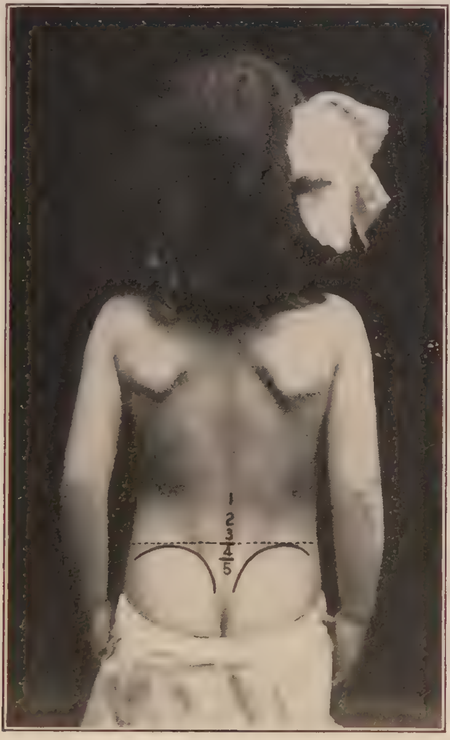

Fig. 87.—Lumbar Puncture. The patient is put near the edge of a table in sitting or lying posture, with the vertebral column strongly arched forward. The puncture is made with a thin, hollow exploratory needle in the lumbar region, in the third or fourth intervertebral space, at a point corresponding to a line drawn between the superior crests of the ilia. (*Sheffield.*)

Lumbar puncture.

Mental State.—In the beginning of the disease the children are usually very irritable. They twitch, grind the teeth, start up with a cry of alarm when disturbed, are annoyed by the least sound in the room, but as the meningitis progresses, or in the tuberculous variety often at its very inception, the patient gradually enters into a state of apathy, stupor, sopor and coma. The experienced clinician in order to arrive at a conclusion rarely needs to wait for the synchronous inauguration of all of the aforementioned symptoms. Indeed, it is quite uncommon to meet with cases which present such an array of typical phenomena. One seldom errs in the diagnosis where persistent vomiting, convulsions, rigidity, photophobia and stupor are grouped together. However, the mere diagnosis of meningitis is not sufficient. It is also the cause and variety we are interested in.

Stupor.

Cerebrospinal Fluid.—With the latest improvements in the technic of examination of the cerebrospinal fluid obtained by lumbar puncture, numerous doubtful points of diagnosis can be cleared up which before the introduction of this diagnostic procedure forever remained a mystery.

Normal cerebrospinal fluid is a clear alkaline fluid, containing but a small proportion of salines, a small quantity of serum globulin, a trace of cholin and a sugar-reducing agent. It is not spontaneously coagulable.

In normal individuals it escapes through the puncture-needle at a low pressure, usually drop by drop. The pressure may accurately be measured by the manometer, but the experienced eye can well appreciate the amount of tension by observing the force of the jet.

The *pressure* is usually increased in divers meningeal irritations and is particularly high in tuberculous and hydrocephalic conditions. As the stream may be altered by the position of the patient, by the viscocity of the fluid, by interference with the flow in its path, etc., the semeiologic importance of pressure is rather slight.

High pressure.

The *color* of the cerebrospinal fluid may be altered by accidental or pathological admixture of blood, pus or pigment. In acute bacterial meningitis the discoloration varies from slight cloudiness to a well-defined purulent turbidity. In tuberculous meningitis the fluid is usually clear or slightly opalescent. The presence of blood is readily recognized and may be due to accidental admixture from the puncture wound or to hemorrhagic pachymeningitis.

Clear fluid in tuberculosis.

The *bacteriologic examination* of the cerebrospinal fluid is of inestimable clinical value since it often furnishes reliable information, not alone as to early diagnosis, but to prognosis and treatment as well. Too much stress cannot be laid upon the fact that in order to obtain conclusive pathologic data the examination of the fluid should be intrusted to one thoroughly experienced in bacteriology and microscopy. Negative results in the majority of instances are due to skepticism and faulty technic. Occasionally repeated examinations are required. Nearly all kinds of micro-organisms have been found. Careful search for the tubercle bacillus should be made in all cases of meningitis, regardless of clinical data. The finding of the tubercle bacillus in the cerebrospinal fluid at a glance settles the diagnosis whereas volumes of descriptions of differential features at best fail. The same applies for the diplococcus intracellularis meningitidis, and other pathogenic bacteria.

Perfect technic.

Detection of bacteria decisive in diagnosis.

For the detection of the micro-organism we may use stained smears (the specimen having been obtained from the coagulum that forms in the fluid on standing or after centrifugation), cultures, or inoculation methods. Where rapid decision is demanded the last two procedures are not adoptable, but as their scientific accuracy is incontestable they are not rarely indispensable in cases of obscure origin and especially in mixed infections.

Cultures.

Cytodiagnosis is based upon the histological study and determination of the number and nature of the formed elements in the cerebrospinal fluid. Normally this fluid contains very few cells, so few that a smear obtained from the deposit after centrifugation only two or three leucocytes may be visible in the microscopic field. The presence of leucocytes in great numbers constitutes anatomical evidence of a meningeal lesion,—namely, of tuberculous nature, where lymphocytes (mononuclears) prevail, and non-tuberculous, where polymorphonuclear leucocytes predominate. This rule applies only to cases which are neither very recent nor very protracted—*i.e.*, to the fully developed acute disease—since lymphocytosis is found also in non-tuberculous meningitis tending to recovery, in acute syphilitic meningitis, and other chronic brain affections; while polynucleosis is occasionally associated with lymphocytosis in chronic tuberculous meningitis.

Mononuclears in tuberculosis.

Of interest *chemically* are the facts that in meningitis the proportion of chlorides in the cerebrospinal fluid is often reduced while that of albumin increased. The albumin consists princi-

Serine.

pally of serine, while normally it is mostly globulin. The fibrin is increased, while the reducing agent is often absent.

Differentiation from typhoid; apex-pneumonia.

Uremia.

Eclampsia.

In the early stages meningitis may be confounded with typhoid fever, pneumonia, acute exanthematous diseases, uremia and eclampsia from other causes. In *typhoid fever* the vomiting is less persistent, diarrhea the rule, impairment of the sensorium less marked and more gradual in development, the spleen enlarged, the fever characteristic (step-curve), and the blood responding to Widal's reaction. *Apex-pneumonia* particularly may be mistaken for acute meningitis. In pneumonia the "cerebral" symptoms usually clear up with the establishment of the signs of pulmonary consolidation, the respiration ratio is increased and expiration is prolonged, and the temperature is evenly high. On the other hand, in meningitis, the nervous symptoms increase with time, respiration is irregular or stertorous and inspiration prolonged and sighing, and the temperature variable. The differentiation between meningitis and a sudden attack of *uremia* is based principally upon the condition of the urine which should always be tested in case of doubt. The history also is very helpful. *Eclampsia* caused by gastrointestinal intoxication, etc., or onset of some febrile disease is apt to be mistaken for cerebral convulsions for the first twenty-four hours only—until the alimentary canal has been cleared, or the other causes of the eclampsia have become apparent.

TUBERCULOUS MENINGITIS.	NON-TUBERCULOUS MENINGITIS.
History: Preceding indisposition.	Apparent good health; infectious disease or otitis.
Temperature: Low in the beginning.	High.
MacEwen's sign: Pronounced.	Slight.
Cerebrospinal fluid: Clear; tubercle bacillus; lymphocytosis (mononuclear).	Cloudy or purulent; no tubercle bacilli; polynucleosis.
The eyes: Optic neuritis; choroid tubercles.	Absent.
Skin eruptions: Indefinite.	Frequently petechiæ.
Paresis: Early.	Late.
Von Pirquet test positive.	Negative.

Remittent fever.

Otitis.

Latent tuberculous meningitis may lead to many errors in the diagnosis. It may be confounded with severe remittent fever, double otitis media (with or without cerebral abscess), syphilitic meningitis, and tumor of the brain. In *remittent fever* the plasmodium malariæ or pigment is readily found in the blood; in *double otitis* examination of the ears reveals local lesions and

Syphilis.

Tumors.

the blood shows marked leucocytosis; in *syphilitic meningitis* there are other evidences of syphilis (choroiditis, rhagades, spirochæte, etc.); in *tumor of the brain* the progress of the disease is slow, and there are focal symptoms (localized paralyses, optic neuritis, etc.) to account for a local lesion. In doubtful cases lumbar puncture and the tuberculin reactions will materially aid in the diagnosis.

Distended fontanelles.

Trough-shaped abdomen.

Hydrocephalic cry.

Coma.

Involvement of cranial nerves.

Hazy corneæ.

The *course of meningitis* varies greatly not only with the cause but with the clinical types of the affection and the severity of the epidemic as well. Some cases are mild and transient, "abortive"; others are extremely malignant, "fulminant," in nature, ending fatally within a day or two, or sooner. The mode of commencement offers no certain indication as to the ultimate course. As previously mentioned primary meningitis begins more suddenly and progresses more rapidly than the secondary variety. The great majority of cases are usually ushered in by profuse vomiting, rise of temperature, severe headache, pain in the back and limbs, sensitiveness of the vertebral column, rigidity and convulsions. The fontanelles are distended, the bowels confined, the abdomen is retracted (trough-shaped) and the urine scanty, often albuminous. During the early period symptoms of excitement of function prevail. The patient is delirious, shrieks (hydrocephalic cry), is very sensitive to noises and light, but very soon he passes into a state of sopor which gradually increases in intensity. At a later period of the disease there is depression of function. The pulse and respiration which in the beginning are accelerated later become irregular and slow, the somnolence deepens to coma, and various paralyses appear. The aforementioned eye-symptoms are usually quite marked and involvement of the facial nerve pronounced. In disease of the base all parts of the facial nerve may be involved; in that of the convexity only the lower part may suffer. In hopeless cases deglutition also becomes affected; the coma increases, the patient can no longer be roused; the conjunctival reflex is abolished, the eyes are smeared with mucus or pus; the corneæ are hazy or ulcerated, the sphincters are paralyzed; and after lingering in this moribund state for another few days the patient is finally relieved of the agony by death. Milder, non-tuberculous, cases may gradually recover. In this event the disease is usually followed by very slow convalescence and frequently by deaf-mutism, aphasia, amaurosis, idiocy, etc. Meningitis sometimes runs a protracted

Chronic course.

course, continuing for weeks with periods of marked improvement, but finally ends fatally. These cases generally represent the chronic form of infantile meningitis, which is essentially a meningoencephalitis.

At best the prognosis is very grave. Tuberculous meningitis is invariably fatal. The mortality in non-tuberculous meningitis ranges between 50 per cent. and 75 per cent. Where operative procedures can be brought into use, *e.g.*, traumatic or otic meningitis with localized lesions, the outcome is more hopeful, provided no time is lost and the patient's general health is fair.

Flexner's serum.

Aside from operative treatment, lumbar puncture for the relief of pressure symptoms, and meningococcic antitoxin, little need be expected from all other methods of treatment in vogue. With the advance in our bacteriologic study of the cerebrospinal fluid and the possibility of early detection of the etiologic factor of the meningitis in question, there is reason to hope that the majority of cases of meningitis will be combated by a curative serum. Wonderful results are already on record from the early use of antimeningococcic serum in meningitis due to the diplococcus intracellularis (Weichselbaum). (See page 95.)

Warm baths.

Iodids.

The symptomatic treatment consists of warm baths every three or four hours; ice-bag to the head, bromids and stronger hypnotics to relieve excessive irritation; small doses of calomel and large doses of sodium iodid; careful nursing (feeding by mouth, gavage[1] or per rectum), and stimulation as necessity arises. Special attention should be paid to cleanliness of the mouth and nasopharynx, and avoidance of decubitus.

Isolation of patient.

When an epidemic prevails all such prophylactic measures should be instituted as are recommended for other contagious and infectious diseases, especial care being taken to disinfect nasopharyngeal discharges.

℞ Natrii iodidi	ʒss	2
Natrii bromidi	ʒj	4
Aquæ menthæ pip.	℥ss	15
Aq. destil.	q. s. ad f℥ij	60

M. Sig.: ʒj every six hours for a child 3 years old. (Routine treatment.)

℞ Hyoscin. hydrobromatis .... gr. $1/500$ to gr. $1/300$.

Sig.: Hypodermically for a child from 3 to 6 years old. (To relieve excessive excitation.)

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[1] *Gavage*, or introduction of food directly into the stomach, is performed in the same manner as lavage (q. v.), except, of course, that the fluid is left in the stomach.

PAROTITIS EPIDEMICA

(Mumps).

acute inf. disease
Char. by swelling of th
parotid
at times
of the
other
salivary glands
micro-unknown -

Primary, idiopathic, epidemic parotitis is a contagious affection of the glandular substance (acini and the ducts) and the interstitial tissue of one or both parotid glands. It most frequently attacks children of from two to twelve years of age, more rarely younger and older ones. (One attack usually confers immunity for life. *Secondary* or *metastatic* parotitis is not rarely met as a complication or sequel of divers infectious diseases and has nothing in common with epidemic parotitis. Infection occurs through the mouth or throat. The specific micro-organism is still unknown.

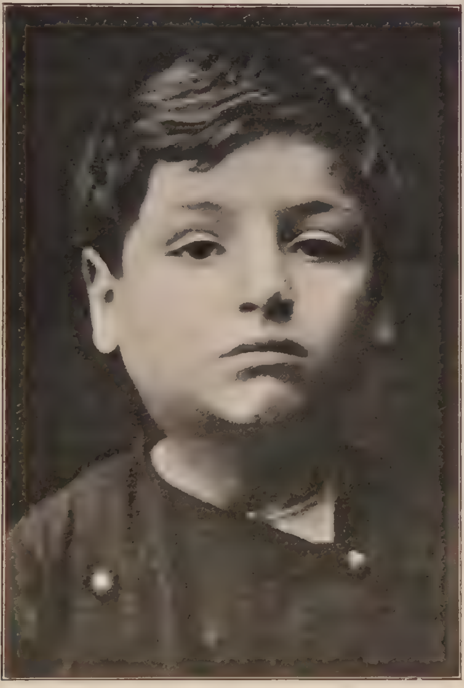

Fig. 88.—Bilateral Epidemic Mumps. (*Sheffield.*)

Differentiation from secondary parotitis.

After an incubation period of from ten to eighteen days and a prodromic stage of about forty-eight hours' duration (marked by general malaise, pain in the region of the ear and throat), typical epidemic parotitis is characterized by a gradually increasing swelling of the parotid gland in front and below the ear and along

Tumefaction.

Involvement of other salivary glands.

the angle of the lower jaw. The swelling increases up to the third or fourth day, remains stationary for another two or three days, and then rapidly subsides. Occasionally the glands undergo suppuration or chronic induration. Quite frequently after subsiding in one parotid the inflammation passes on to the other; more rarely both parotids are involved simultaneously. The overlying skin is usually colorless; more rarely pale red, glistening and painful. The inflammation may extend to the other salivary glands, or to the lymph and lacrimal glands, involve the tonsils, lids, conjunctiva, and less frequently the testicles, or ovaries, vulva or breast—usually on the same side as the parotid affected. Occasionally the submaxillary glands alone are involved, and, where the parotitis is bilateral and severe, there may be a confluence of the bilateral tumors.

Testicles.

Ovaries.

Complications.

Isolation of patient.

Except pain in swallowing, opening of the mouth, chewing, turning the head, etc., headache, occasionally vomiting, and a rise of temperature during the first or second day of the disease, the patient usually suffers no discomfort. Of course, the symptoms are materially changed if the testicles (orchitis parotidea) or ovaries, etc., are involved, or if complications make their appearance—rather rarely to be observed in cases of ordinary severity. Otitis and nephritis form the most frequent complications. They may occur during convalescence, less often during the acme of the disease. The nephritis is usually hemorrhagic, but benign, in nature. The otitis not rarely leads to deafness. Other complications of parotitis on record are: Meningitis, encephalitis, divers paralyses, psychoses, pericarditis, endocarditis, arthritis, etc.—the same as are apt to be met in many other acute contagious and infectious diseases. Notwithstanding the possibility of grave complications and sequelæ, the prognosis of parotitis is almost always favorable, rarely calling for any elaborate therapeutic measures. A few days' rest in bed, fluid diet, the salicylates for the relief of pain, and local application of lead- or potassium-iodide ointment with or without 10 per cent. of ichthyol, usually suffice to effect a cure in the majority of uncomplicated cases. Complications should be treated according to indications. It is advisable to isolate the patient for about three weeks.

Parotitis may be mistaken for swellings in the same region, resulting from stomatitis, alveolar periostitis, retropharyngeal abscess, and infected glands from other causes. Bearing in mind the cause, consistency and location of the tumor; the presence or absence of an epidemic, and the course and duration of the disease, there ought not to be any great difficulty in arriving at a correct diagnosis.

The course of secondary parotitis differs with its cause.

PERTUSSIS

(Tussis Convulsiva, Whooping-cough).

Infectious d. char. by cat. infl
of air-passages + peculiar parox
of cough ending in a
loud whoop
inspiration

Whooping-cough is a highly communicable, epidemic and sporadic affection, during its height characterized by sudden more or less frequent paroxysms of coughing which from time to time are interrupted by deep, stridulous inspiration, and followed by a period of apparent euphoria of variable duration. The specific germ of the disease is still unknown. As a rule, the course of pertussis is divisible in three distinct stages: Stadium catarrhale, convulsivum, and decrementi.

The *stadium catarrhale,* which lasts about ten days, begins after an incubation period of from five to nineteen days. It is sometimes preceded by a few indefinite prodromata, consisting of loss of appetite, languor, restless sleep, and slight fever, and as these symptoms gradually disappear they become replaced by those of a simple catarrh of the upper air-passages, so that the advent of the grip or measles is often suspected. At first the cough is short, hacking, sometimes croupy in character, but steadily it grows worse, though returning at longer intervals. It is especially troublesome at night, and what, as a rule, is particularly characteristic of the whooping-cough, the cough fails to respond to the remedies usually efficient in ordinary "coughs and colds." Toward the end of the catarrhal stage the child is off and on attacked by a paroxysmal cough, thus indicating the early advent of the second, convulsive stage of the affection.

Catarrhal stage.

Short, croupy cough.

The *stadium convulsivum* may last from two to four weeks or, if left to run at random, as many months. The cough is violent and explosive, each paroxysm being often preceded by a slight aura, by vomiting, sneezing, etc., so that older children are usually aware of its approach.

Paroxysmal stage.

Violent, explosive cough.

Children able to walk usually run toward a person or object to support themselves during the attack, and infants manifest the approach of the paroxysm by a sudden outburst of crying. Each paroxysm, which lasts from a half to five minutes, consists of a number of short, barking, expiratory acts of coughing, from time to time interrupted by deep whistling or stridulous inspirations—which constitute the "crow" or "whoop"—and is ordinarily (may be followed by a second or third fit of coughing) concluded with the expulsion of glassy, tenacious mucus and often also by vomiting of food residue. During a paroxysm the face is at first red, then cyanosed, and the veins in the neck swell. As the attacks grow worse, there is considerable venous stasis, puffiness of the face (which remains occasionally permanent), especially at the eyelids; there is bleeding from the nose and throat, in the skin, conjunctiva, more rarely from the ear (rupture of the drum-membrane, which heals spontaneously), in the meninges (cause of convulsions), etc. In delicate and young children a paroxysm is not rarely associated with involuntary defecation and urination, and at times also general convulsions. The number of paroxysms varies between ten and sixty in twenty-four hours. (They are more frequent with the patient living in unhygienic surroundings, after overloading of the stomach, on excitement from any cause (crying, laughing, etc.), irritation of the nasopharynx and larynx (often a useful means of diagnosis!). In mild and moderately severe cases the child is apparently quite well between the attacks; in very severe cases, however, the patient is weak, pale, emaciated and suffering from troublesome bronchitis and often from a number of other grave complications soon to be related. Under proper treatment the paroxysms in uncomplicated cases are, as a rule, more or less checked after from ten to twenty days. The paroxysmal stage is then followed by the regressive stage, *stadium decrementi*. The attacks become less frequent, they lose their typical character, the cough returns to the original catarrhal type and finally abates entirely. This declining stage ordinarily lasts for from two to three weeks. Occasionally, however, especially in cases exposed to unsanitary conditions and careless treatment, this stage may continue for months and be interrupted by relapses which often undermine the patient's constitution and lead to irreparable lesions in different organs of the body.)

Whoop.

Venous stasis.

Convulsions.

Declining stage.

Divers complications and sequelæ have been noted: *Of the lungs:* Capillary bronchitis, bronchopneumonia, emphysema, and bronchiectasis, phthisis, and acute miliary tuberculosis (as a result of caseation of the bronchial glands); *of the heart:* dilatation, pericarditis and myocarditis; *of the brain:* divers paralyses (hemiplegia, facial, laryngeal, etc.), hemorrhagic, or tuberculous meningitis, encephalitis, softening of the brain, mental

Grave complications.

affections, such as imbecility, idiocy, and different forms of insanity; *of the spinal cord:* myelitis, poliomyelitis, hemorrhagic inflammations, and polyneuritis; *of the ears:* otitides, with or without permanent deafness; *of the eyes:* amblyopia, amaurosis; also nephritis, sublingual ulceration (as a result of friction of the sublingual parts against the teeth during a paroxysm), severe epistaxis, and emphysema cutis from rupture of some pulmonary alveoli. Delicate, especially bottle-fed babies not rarely suffer from gastroenteritis with subsequent marasmus, and, finally, sudden collapse from respiratory and heart-failure may ensue at the acme of a protracted fit of coughing.

Heart-failure.

Fortunately the cases are not all of so grave a nature and so dreadful in their consequences. Numerous abortive cases are on record in which the second stage is devoid of the "whoop" (sometimes replaced by attacks of sneezing), and the third is of very brief duration, so that in the absence of an epidemic or a definite source of infection there is justification for a doubtful diagnosis. When the whoop is absent some assistance in the diagnosis may be obtained by a careful examination of the blood, which will show that during the second stage the polynuclear cells are increased twice in number, and the lymphocytes about four times. Of diagnostic importance also is the fact that the urine has a high specific gravity (1022 to 1032) and contains an excessive amount of uric acid crystals. The diagnosis is often almost impossible during the first stage of the affection, especially if following—which is quite frequently the case—measles, and time alone is the only reliable guide.

Mild form.

Characteristic blood changes.

Severity generally under-estimated.

No other communicable affection of childhood is as lightly regarded by the laity and as carelessly treated by the physician as that under discussion. Notwithstanding the facts that it prevails during the greater part of the year; that its mortality ranges between 4 per cent. and 6 per cent. as an immediate result of the disease, and at least as high as 10 per cent. in consequence of complications and sequelæ—thus demanding a greater proportion of victims than typhoid and pneumonia combined—no strenuous effort is being made to still its ravages, to arrest its spread, or to abort its course. The fallacious impression has gained firm ground that whooping-cough "must run its course of from six to eighteen weeks," and even the scientific, practical physician wisely nods his head in affirmation and despair, lest he be ridiculed by the therapeutic nihilist. One has to be bold to venture to claim

Prompt treatment very helpful.

success in allaying the spasm, reducing the number of paroxysms, and preventing the dreadful complications of the disease, and the one who dares to proclaim the possibility of cutting short the lengthy course courts everlasting infamy! All the same, the severest attack of whooping-cough properly treated may be rendered almost innocuous, or at least free from grave consequences.

As soon as the diagnosis has been established with any fair degree of certainty (even earlier where direct infection is demonstrable), the patient should be isolated, and the expectoration disinfected. For the latter purpose a sputum cup is very helpful. Isolation should be practised principally during the expectorating period—at least three weeks.

Fresh air.

Fresh air being one of the most essential and efficient therapeutic measures, the child should be kept outdoors the greater part of the day (except in the presence of grave complications), and the rooms constantly aired with the patient indoors. Whenever possible, two or more rooms should be made use of. The food should be bland and strengthening, and given in small amounts preferably after the paroxysms. The clothing should correspond with the season of the year. It is true we possess no ideal specific cure against the disease, but a great deal can be done to lessen the number and severity of the paroxysms and to prevent complications by resorting to the following medicinal agents:—

℞ Olei eucalypti	ʒiv	15
Tinct. benzoini comp.	q. s. ad f℥ij	60

M. Sig.: ʒj in a pint of hot water, to be used for inhalation through a croup kettle three times a day.

Quinine.

℞ Quinine ethyl carbonate,[1] or diquinine carbonic ester[2]	ʒss	2
Syr. simplicis	q. s. ad f℥ij	60

M. Sig.: ʒj every two to four hours, according to the severity of the paroxysms, for a child 3 years old.

Antipyrine.

Whenever necessary a small dose of some morphine preparation with or without 2 grains of antipyrine may be administered to induce rest or sleep, and, where the heart is weak, a fresh infusion of digitalis will prove a grateful addition. Numerous other proprietary remedies have been found serviceable, but caution is commended in their promiscuous use.

The paroxysms may frequently be controlled by pulling the lower jaw downward and forward. This manipulation is harmless and painless. Its application is contraindicated only in the presence of food in the mouth or esophagus.

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[1] Euquinin. [2] Aristochin. Because of their tastelessness these preparations are to be preferred to ordinary, bitter, quinine.

Chloroform.

Chloroform anesthesia will sometimes relieve the paroxysms almost magically, and should be tried in desperate cases, especially in those associated with convulsive seizures.

Complications and sequelæ arising should be treated according to indications.

Antispasmodics.

℞ Ext. belladonnæ fl.	gtt. iv	0.25
Vini ipecacuanhæ	gtt. xvj	1
Natrii bromidi	gr. viij	0.5
Syr. amygdal. q. s. ad	℥ij	60

M. Sig.: ʒj every two to four hours for a child 2 years old.

℞ Creosoti carbonatis	ʒiv	15

Sig.: Gtt. ij in a teaspoonful of sweetened water every three hours for a child 3 years old.

Infectious d. T. B. bacillus

Acute

TUBERCULOSIS.

INTRODUCTORY REMARKS.

Without denying the possibility of ante-natal direct bacillary transmission of tuberculous disease from parents to offspring, it may be set down as absolutely certain that, with but very few exceptions, tuberculosis in infancy and childhood, as in adolescence, is acquired as a result of post-natal infection by the tubercle bacillus of Koch. The bacillus invades the human organism principally through the respiratory (by inhalation) and alimentary (by ingestion) tracts, and less frequently through the skin or mucous membranes (slight traumatism, skin eruption, etc.).

Acquired affection, by inhalation and ingestion of tubercle bacillus.

(The readiness with which infection occurs depends chiefly upon the power of resistance of the patient and the environment in which the patient is forced to live.) This explains the greater frequency of tuberculous disease in children of tuberculous parentage. An undermined constitution from one cause or another forms an easy prey to the tuberculous germ and, varying with the primary seat of infection, the natural recuperative strength of the tissues involved and the therapeutic measures adopted to resist and combat further systemic invasion, tuberculous disease may remain localized or become general, and pursue an acute or chronic course.

Undermined constitution.

(The successful management of tuberculosis rests upon a thorough appreciation of the aforementioned facts.) We possess no specific remedy against tuberculosis, once fully established, but the disease is certainly preventable and in its incipient stage curable—a great deal more than can be said of a number of non-tuberculous, organic affections.

Prophylaxis.

Outdoor air.

Free breathing.

Prevention of tuberculosis in a child must begin immediately after its birth. The air the infant is to breathe should be pure, the room it is kept in sanitary and well ventilated, though warm enough to suit its needs. From earliest infancy the child should be gradually accustomed to outdoor air, and, as it grows older, it should spend most of the day outdoors, except when the weather is particularly bad. In this event it should remain well dressed in front of an open window. Especial attention should be paid to its breathing. Any obstruction to free nasal breathing, be it adenoids, hypertrophy of the tonsils or of the nasal mucous membrane or deformity of the nasal bones, should be treated or removed without delay. The child should be taught to breathe deeply—few children know how to breathe, as is readily evinced in examining a child's chest. Infants should be encouraged to cry off and on, and older children to recite and sing in the open air. As the child grows old enough intelligently to follow instructions, it should be taught the following breathing exercises :—

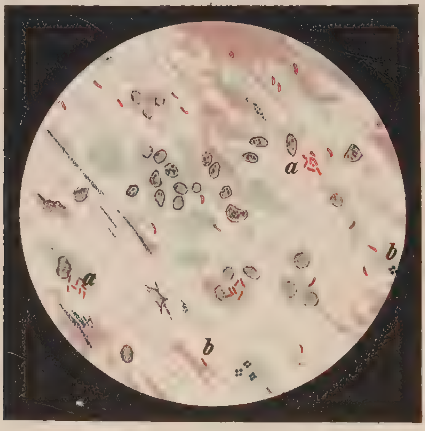


Fig. 89.—Tubercle Bacilli and Micrococcus Tetragenus (sputum). Gabbet's stain, Leitz ocular I, oil immersion 1/12. (*a*) tubercle bacilli; (*b*) micrococcus tetragenus. (*Lenhartz* and *Brooks.*)

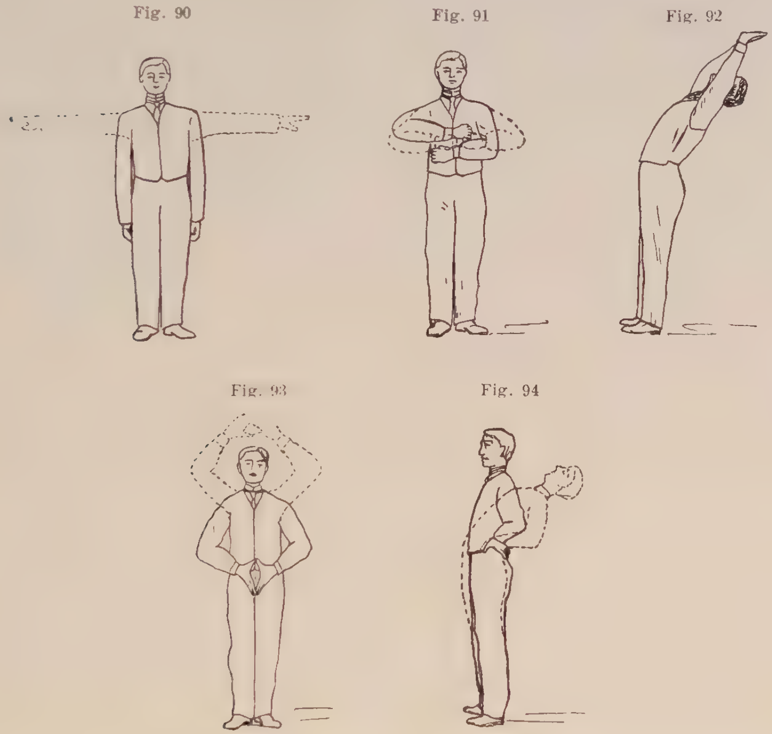
1. Deep inhalation, while raising the arms to a horizontal position; slow exhalation, bringing the arms down. (See Fig. 90.)

2. Deep inhalation with the arms placed lightly upon the

front of lower portion of chest; slow exhalation, bringing the arms down. (See Fig. 91.)

Breathing exercises.

3. Deep inhalation, while bringing the arms first to horizontal postion then above the head, and lastly—while still holding the breath—bending the upper body backward; slow exhalation, while lowering arms sideways. (See Fig. 92.)



Figs. 90 to 94.—Breathing Exercises. (*Sheffield.*)

4. Deep inhalation, while bringing the hands together in front of abdomen, and from here slowly along the thorax and chin above the head and as far as possible in back of it; slow exhalation, bringing the hands down to original position. (See Fig. 93.)

5. Deep inhalation, while bending the upper body as far back as possible, with the hands fixed on the hips; slow exhalation, while resuming original position. (See Fig. 94.)

Mouth closed.

During the breathing exercises the child assumes the position of military "attention." He breathes with the mouth closed, occupying about four seconds for inhalation, four seconds for retention of the air and three seconds for exhalation. The exercises should be practised either outdoors or in front of an open window, at first four or five times a day, but, after the child gets accustomed to properly expand his chest during the respiratory act, only once or twice a day or not at all. The breathing exercises, like any other physical work, should not be overdone, and never continued so long as to become tiring. As prolonged holding of the breath interferes with the normal heart's action, it is contraindicated in organic heart disease. Short-distance running, and peaceful outdoor games (handball, basketball, and tennis) also are helpful to expand the lungs. The principal benefit derived from these breathing exercises is the purification of the lung tissue by the free inflow and uniform distribution of oxygen, thus preventing pulmonary congestion which acts as a predisposing cause of tuberculous infection.

Moderation.

What pure air does for the prevention of pulmonary tuberculosis, suitable feeding from birth will do for the prevention of tuberculosis of the alimentary tract. It is highly essential ever to bear in mind that tubercle bacilli rarely, if ever, survive the action of normal digestive juices. The gastroenteric tract, especially the stomach, therefore, should be spared pathologic alteration. Breast milk of a healthy mother or wet-nurse should at all times be the food of choice for an infant up to nine months old. With increasing age the dietary should undergo gradual changes, always selecting, however, such articles of food as will best accomplish the object in view, *i.e.*, ample nutrition for the growth and development of the child with least possible injury to the digestive organs. Overfeeding especially is to be avoided. It goes without saying that contaminated food should never form a part of the dietary. Cows' milk of doubtful purity should be sterilized, and other articles of food of such character boiled. The teeth should receive especial attention, as cavities of decayed teeth not rarely harbor tubercle bacilli and early loss of the permanent teeth forms one of the principal causes of acute and chronic dyspepsia—as a result of insufficient mastication of the food—and indirectly enhances the development of tuberculosis. Children should be taught to eat slowly, and to refrain from eating between meals. For further details as to mode of feeding, see page 60.

Ample nutrition.

Germ-free cows' milk.

Tuberculous infection through the skin and contiguous mucous membranes should be guarded against by scrupulous cleanliness of these structures, avoidance of external injury and skin eruptions, and by immediate treatment of open wounds and all such skin lesions as are associated with itching and compel scratching. Those intrusted with the care of babies and older children should be instructed to give their charges a tub bath (see page 83) in the evening and a sponge bath in the morning followed by gentle rubbing of the entire body. Of course, the bathing should include careful cleansing of the nails, which should be kept clipped short; of the ears, of the nose and scalp, and, in older children, also of the teeth. From earliest infancy children should very gradually get accustomed to cool sponge baths. These are very beneficial to counteract the susceptibility to frequent colds. At first the infant may be given a cool alcohol sponge, and after toleration has been established the alcohol should gradually be replaced by water, and finally by full cool tub- or shower-baths. The advisability of cleansing the infant's mouth is still a matter of great difference of opinion. I am inclined to favor gentle *wiping* of the infant's mouth twice daily with a cotton swab dipped in sterile water. Older children should be taught the use of a soft brush for the teeth and an antiseptic gargle for the mouth and throat. The importance of early removal of nasopharyngeal obstruction to breathing has already been alluded to. This question cannot too strongly be emphasized, for the adenoid tissue in addition to interfering with free respiration is surely one of the most rampant sources of tuberculous infection. Skin eruptions should at once be combated. This refers especially to running sores from whatever cause, and to all skin diseases which sooner or later lead to maceration and denudation of the skin. Intertrigo in infants is best prevented by frequent changing of the diapers and keeping the buttocks perfectly clean and dry. The child should be kept from scratching the affected portions of the skin by immediate application of antipruritic drugs and by restraining the child's hands by means of one of the many useful contrivances. Open wounds should be dressed antiseptically until healed. Vaccination wounds especially should receive careful attention. Certain though it be that latent tuberculosis is occasionally lighted up through vaccination, and that tuberculosis has in very exceptional instances been traced to vaccine infected by tubercle bacilli, it is absolutely settled that the great majority of cases of tuberculosis following vaccination are the result of direct bacillary infection through an unprotected vaccination wound.

Free nasopharynx.

Attention to open wounds.

Care of vaccination wound.

Effective as these local measures are in the prevention of tuberculosis, their efficiency is very insignificant as compared with the natural defensive resources of a healthy constitution. Our aim, therefore, should be directed chiefly, from earliest infancy, to render the patient, so to say, immune against tuberculosis. This is best accomplished by outdoor life, wholesome nutrition, and sanitary environment. Those showing a tendency to remain delicate in health should reside in the country.

Fostering immunity.

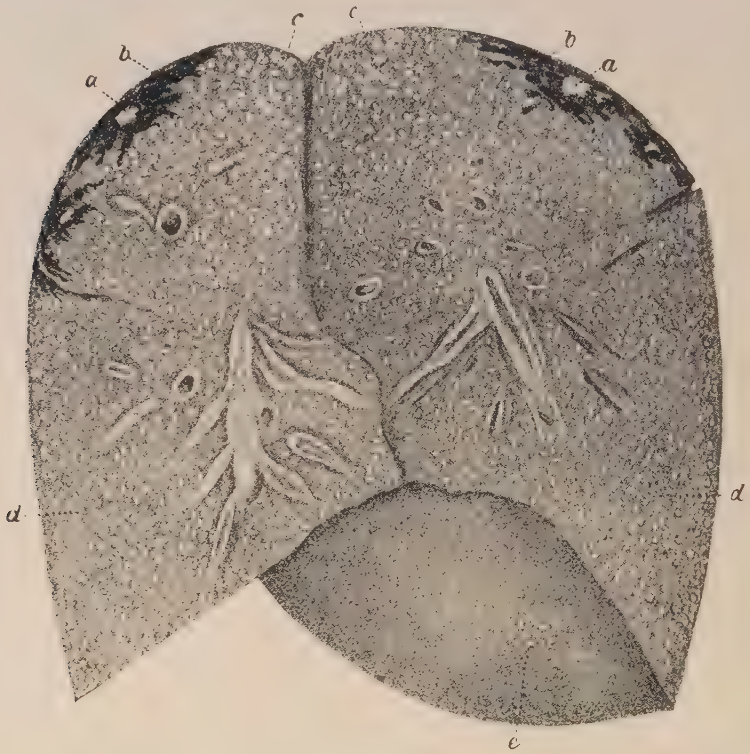


Fig. 95.—Acute Pulmonary Miliary Tuberculosis (Cut Surface of the Lung). (*a*) So-called obsolete tubercle (old encapsulated caseous focus. (*b*) Induration. (*c*) Caseous, partly agminated nodules (transverse section of caseous bronchi). (*d*) Submiliary non-caseated tubercle in the true lung tissue. (*e*) Tubercle of the pulmonary pleura. One-half natural size. (*Langerhans.*)

MILIARY TUBERCULOSIS
(Hasty Consumption).

Wide distribution of lesions.

This disease is characterized by wide distribution of the tuberculous lesions. The latter are from a pinhead to millet-seed in size, gray or yellow in color, and firm in consistence. They are found scattered throughout almost all organs and tissues of the body, but especially the lungs and bronchial glands, intestines and mesenteric glands, the liver, spleen, kidneys and bladder, and the brain and its coverings. They may remain latent for some time, or give rise to indefinite symptoms, such as anorexia, dyspepsia, gastroenteritis, and emaciation, or symptoms of pulmonary phthisis. (The outbreak is often determined by some intercurrent disease or traumatism, but once established it usually runs a very violent course.)

Latent course.

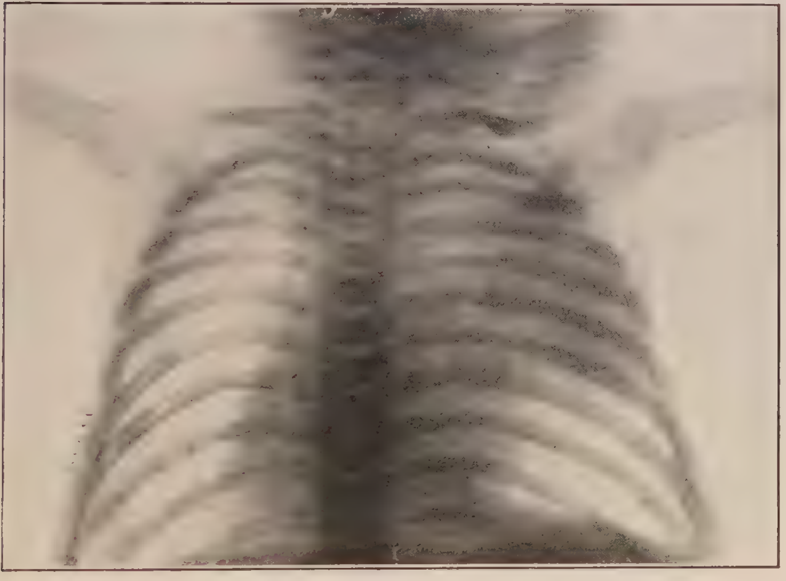


Fig. 96.—Miliary Tuberculosis (skiagram). (*Sheffield.*)

The temperature rises, is intermittent, hectic in character, only rarely drops to normal, and may be associated with chills and sweats. In the beginning, especially in the absence of marked pulmonary symptoms, and in the presence of large liver or spleen or both, the disease greatly resembles malarial fever or typhoid. (Careful examination, however, reveals the absence of the malarial or typhoidal germs in the blood.) Where signs of pulmonary disease predominate, it is readily confounded with lobar or lobular pneumonia. In such cases the diagnosis is extremely difficult and often can be decided only by microscopic examination of the

Hectic fever.

Resemblance to malaria, typhoid and pneumonia.

sputum (frequently negative) and the tuberculin test. As the disease advances the diagnosis may be based upon the extreme emaciation, multifariousness of the symptomatology, and the violence and persistence of the febrile attacks.

Pulmonary, intestinal and cerebral symptoms.

The symptoms and course of the disease differ with the seat of the lesions. The lungs almost invariably show signs of consolidation (dullness, crepitant râles, dyspnea, cyanosis, short cough), and the intestines rarely escape involvement. In some cases brain symptoms (apathy, jactitations, stupor, localized convulsions, tubercles in the choroid, etc., up to a typical picture of meningitis) predominate; in others again symptoms of disturbed circulation (marked cyanosis, edema, rapid feeble pulse, anemia and exhaustion, etc.) prevail. The latter phenomena usually precede the fatal issue, which generally occurs within from four to eight weeks. Cases running a subacute course may last a few weeks or months longer, are not rarely erroneously diagnosticated and treated as marasmus, their true nature not being detected until post mortem. It is in those cases, particularly, that the von Pirquet or Calmette reactions are so helpful in the diagnosis, and should always be resorted to early. For then and then only may our efforts to arrest or possibly cure the disease prove successful.

Differentiation from marasmus.

For details of treatment see page 363.

PHTHISIS PULMONUM

(Tuberculosis of the Lungs and Bronchial Glands).

The lungs proper, the bronchial glands, or both, may be the primary seat of tuberculous deposits. The upper lobes are more frequently affected than the lower, and the portions adjacent to the bronchial glands more so than the remaining parts. The pathologic changes consist essentially in the formation of variously sized caseous nodules filled with colonies of tubercle bacilli and large, so-called giant cells, and subsequent softening and breaking down of the nodules, forming cavities which may vary in size from a pea to a walnut or larger. In some cases, especially in those receiving early and suitable treatment, the tuberculous process is arrested by encapsulation of the necrosed structures by newly formed connective tissue, leading to contraction and formation of a firm cicatrix. In this event the enclosed caseous masses are in part absorbed, and in part calcified.

Pathologic changes.

The tuberculous affection of the bronchial glands also consists

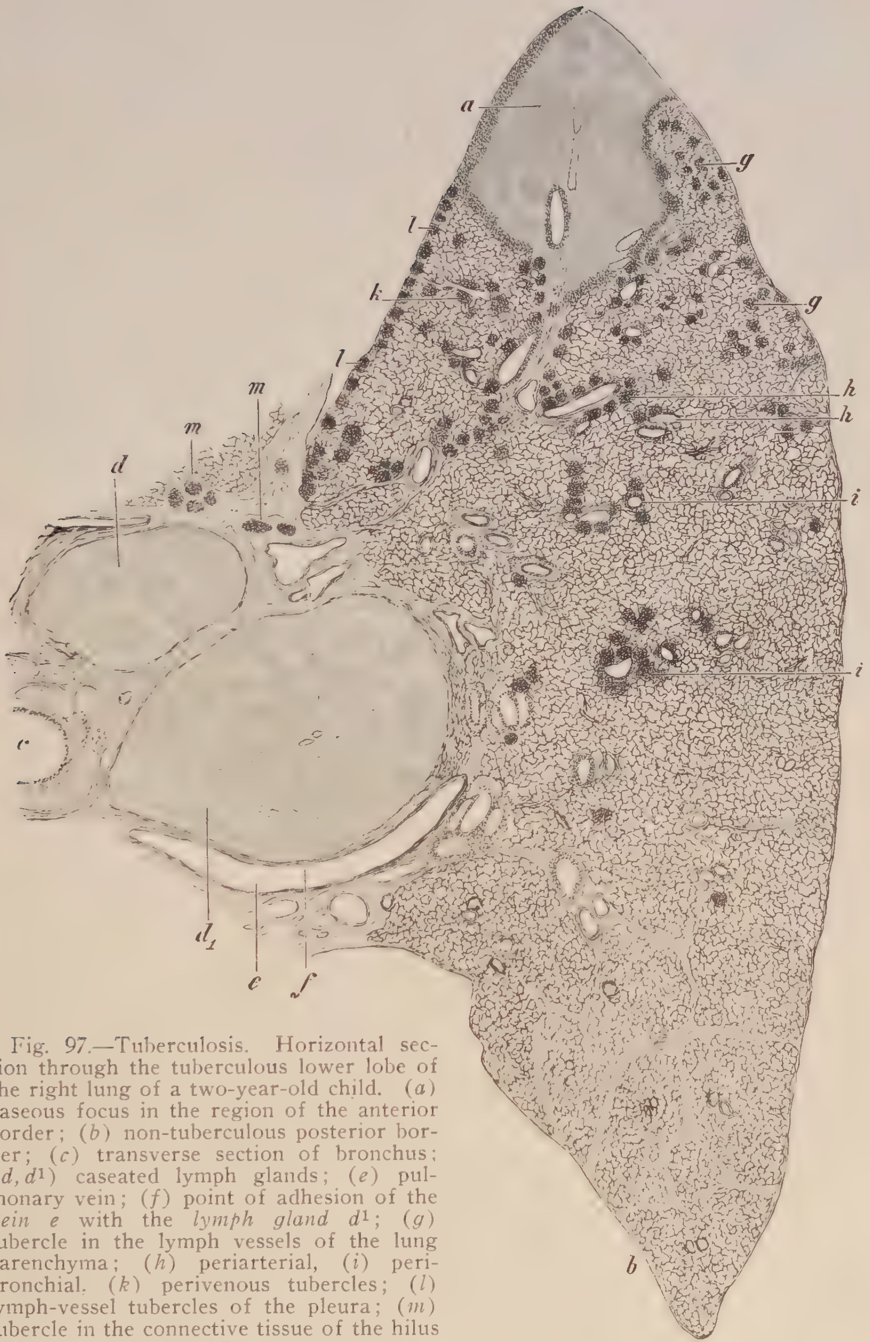


Fig. 97.—Tuberculosis. Horizontal section through the tuberculous lower lobe of the right lung of a two-year-old child. (*a*) caseous focus in the region of the anterior border; (*b*) non-tuberculous posterior border; (*c*) transverse section of bronchus; (*d*, *d*¹) caseated lymph glands; (*e*) pulmonary vein; (*f*) point of adhesion of the *vein e* with the *lymph gland d*¹; (*g*) tubercle in the lymph vessels of the lung parenchyma; (*h*) periarterial, (*i*) peribronchial, (*k*) perivenous tubercles; (*l*) lymph-vessel tubercles of the pleura; (*m*) tubercle in the connective tissue of the hilus of the lung. × 3. (*Ziegler.*)

Hyperplasia of bronchial glands.

in hyperplasia and caseous degeneration. This process usually (sooner or later) extends to the contiguous structures, exerts pressure upon the adjacent blood-vessels, nerves, and bronchi, and, after forming adhesions, may displace, erode and perforate these parts. In this manner not only may tuberculous infection be rapidly carried throughout the lungs and more distant organs (producing an acute or chronic tuberculous pneumonia), but perforation of a blood-vessel or bronchus or entrance of caseous masses into the trachea may unexpectedly produce sudden and often fatal hemorrhage or suffocation.

Emaciation.

Paroxysmal cough.

Diagnosis based upon entire clinical picture.

Tuberculous sputum.

The symptoms vary with the primary seat of the lesion and the subsequent pathologic changes. A small tuberculous focus, be it in the lung or bronchial glands, rarely gives rise to any definite clinical phenomena. As a rule, in the beginning the disease pursues a latent course. This is especially true in infants. The child is pale, loses in weight, often notwithstanding good appetite; gets tired on slightest exertion, "hems" and coughs a little, and the temperature rises somewhat in the evening. Sooner or later the symptoms become more distinct. Emaciation, cough, and gastrointestinal disturbances increase in severity, the child suffers from dyspnea, and, if the bronchial glands are involved, from paroxysmal attacks of cough, greatly resembling pertussis. (This cough is the result of pressure exerted by the enlarged bronchial glands upon the pneumogastric and recurrens nerves.) Physical signs, however, are often still wanting. Occasionally percussion over the mediastinum may reveal increased dullness, but in infants this symptom is not pathognomonic in view of the physiologically large thymus. Indeed, the disease is often not detected until grave, not rarely fatal, symptoms (*e.g.*, hemoptysis, hectic fever) announce the seriousness of the condition. The diagnosis of pulmonary phthisis in infants, therefore, must be based upon the entire clinical picture, rather than the local symptoms. If, for example, bronchial catarrh is associated with progressive emaciation, multiple glandular swellings, protracted diarrhea and possibly also some bone or joint disease, the diagnosis of tuberculosis is justifiable even though careful examination of the thorax fails to disclose pulmonary consolidation or cavity. For corroborative evidence we should carefully examine the child's sputum (obtained by means of a catheter introduced to the base of the tongue) for tubercle bacilli, and employ the tuberculin test.

In older children the symptomatology of pulmonary tuberculosis is essentially the same as in adults. Its onset is usually insidious, and quite frequently follows delayed convalescence from some acute disease, such as pertussis, morbilli, broncho- or lobar pneumonia and the like. The child fails fully to recuperate, is pale, thin, and feeble; suffers from slight shortness of breath, dry cough, chilliness and fever. At first these symptoms are more or less masked, but as the lung destruction advances the symptoms and physical signs rapidly grow worse. The cough becomes persistent, often distressing, especially at night, and attended by expectoration and pain. The fever is intermittent or remittent (hectic) in character. It is usually normal or slightly above normal in the morning, and from two to three degrees higher in the evening. It is often preceded by chilliness and followed by profuse sweating. During the height of the fever the cheeks are usually brightly flushed and contrast strongly with the remaining portions of the face, which are deathly pale. Night-sweats are often observed early in the course of the disease. With further progress of the disease, the expectoration becomes mucopurulent or purulent, mummular, and streaked with blood; the fever more irregular, and attended by great exhaustion, and the emaciation profound.

Irregular fever.

Night-sweats.

The agony may further be aggravated by the concurrence of a number of painful complications. The disease may extend to the pleura (pleuritis sicca or with serous or hemorrhagic effusion); to the trachea and larynx (dysphagia, frequent hemorrhages, and aphonia); to the alimentary tract (colliquative diarrhea); and where the bronchial glands or pleura are involved, to the pericardium (pericarditis). By this time and sometimes at an earlier period the child presents a characteristic, ghastly appearance. The cheeks are hollow, the eyes and temples sunken, the bones of the face and the ears prominent, the nose is pointed and drawn, and the hair thinned, lusterless and brittle. The face is either deathly pale or marked by florid redness along the zygomatic regions. The neck is wasted, the supra- and subclavicular spaces are depressed, the shoulders stoop, and the shoulder blades project wing-like far beyond the shrunken, immovable spine. The thorax is narrow and contracted, and the ribs overlap each other, effacing the intercostal spaces. The abdomen is flat or deeply sunken below the strikingly prominent pelvic bones. The extremities are mere skin and bone and their

Pleurisy.

Dysphagia.

Hemoptysis.

Characteristic facies.

Contracted thorax.

epiphyseal ends seem greatly enlarged as they protrude through the wasted, arid integument.)

The physical signs vary with the stage, location and extent of the lesions. As already mentioned tuberculosis of the bronchial glands may by physical examination entirely escape observation. The same holds true of cases where the tubercles are scattered throughout the lungs and do not coalesce. On the other hand, where pulmonary consolidation (tuberculous pneumonia) occurs early and progresses rapidly, the physical signs resemble those of ordinary pneumonia, *i.e.,* dullness on percussion, prolonged expiration, increased vocal fremitus; fine, coarse and crepitant râles, and bronchial breathing. To these may be added the physical signs of dry or serohemorrhagic pleurisy (see page 275), which frequently accompanies phthisis pulmonalis. Where cavities are formed, the physical signs consist of cavernous respiration, bronchophony or pectoriloquy. The percussion resonance is amphoric, if the walls around the cavity are thin and tense; cracked-pot sound, if the walls are thin and relaxed; and dull, if the walls are thick. If pneumothorax is present, the percussion sound is tympanitic, and the respiratory murmur is lost; while hydropneumothorax gives rise to tympanitic resonance above water line, dullness below, and metallic tinkling on auscultation.

Pulmonary consolidation.

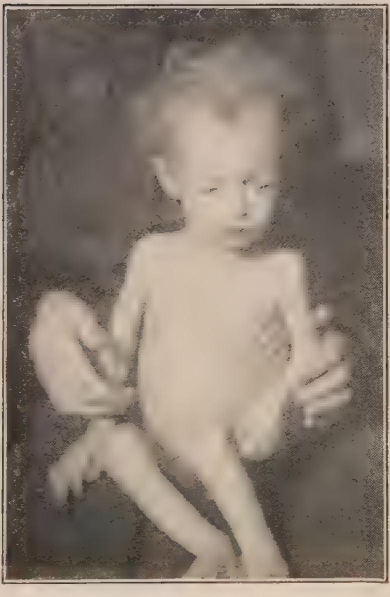


Fig. 98.—Phthisis Pulmonum (child 20 months old). (*Sheffield.*)

Cavernous breathing.

Cracked-pot sound.

The poignancy of the clinical picture just depicted notwithstanding, errors of diagnosis are quite possible. Pulmonary phthisis may readily be confounded with bronchial dilatation, localized empyema, fetid bronchitis, pulmonary gangrene and

Differential diagnosis.

syphilis. In view of the prognostic importance of an early diagnosis of tuberculosis, it is imperative to employ every means of diagnosis (especially repeated examination of the sputum, and the tuberculin reaction) to clear up all doubt.

The course and duration of phthisis pulmonum ranges within very wide limits. Not only is it true that tuberculosis may proceed a latent course for months or years and suddenly break out —often after some trivial cause, such as vaccination, measles, etc.—and rapidly end fatally under symptoms of lobular or lobar pneumonia and the like, but post-mortem examinations have repeatedly established the fact that after existing for some time, with or without indications of their presence, tuberculous lesions may heal spontaneously never to return. As a rule, however, pulmonary phthisis in young children runs quite an acute course. Unless the disease is arrested in its incipiency, infants usually succumb to it within from four to eight weeks, either from the immediate effects of the pulmonary lesions or as a result of generalized tuberculosis not rarely of the miliary variety. In older children the disease pursues a less violent course, and, as in adults, shows a tendency to remain localized at its originally infected focus until a very late stage of the disease. If the tuberculous process is allowed to continue, death invariably occurs in from two to three years or earlier—either from asthenia (with symptoms of gradual exhaustion, profound anemia, dropsy, etc.) or from apnea (suffocation by sudden hemorrhage, rupture of large cavity, pulmonary edema, etc.). On the other hand, if the tuberculous process is detected in its *incipiency*—which is quite possible with the existing modern diagnostic methods—and immediately and energetically treated, the chances for arrest and eventual cure of consumption of the lungs are very good indeed.

Exceptionally spontaneous healing.

The treatment comprises outdoor life, good food, personal hygiene, and symptomatic medication. Whenever possible, tuberculous children should be sent to country regions where the climate is dry and of equable temperature, so as to allow the patients to enjoy outdoor air the greater part of the day. The climates of New Mexico, Arizona, and Egypt are best suited for the purpose, although a great many patients will be found to do well in Colorado, in the Adirondacks and Sullivan County of New York, in Montana, Wyoming and North Carolina. Those financially incapacitated to take advantage of these climates should be removed to climatically less favorable mountain regions

Outdoor life.

or even to ordinary city suburbs, but at all events should not be left to perish in overcrowded, unsanitary tenement districts. It is often of great advantage to place the child in an up-to-date sanitarium—if possible in a private room—as the principles of the treatment are more accurately enforced (and with less resistance on the part of the patient) under the supervision of a reliable physician and nurse of a properly conducted sanitarium, than at the patient's residence among his timid and sympathetic immediate relatives.

Sanitarium treatment.

The diet should vary with the age of the patient, but should be highly nutritious and liberal. Milk, meat, eggs, fresh fish, oatmeal, peas, beans and lentils, carrots, spinach, asparagus, potatoes, etc., in addition to an ample supply of bread and butter, should form the principal components of the regular meals. Between meals the child should receive plenty of fresh fruit or fruit juices, and, to satisfy its craving for condiments, a small portion of milk chocolate or calf's foot jelly.

Highly nutritious diet.

The room occupied by the patient should be large and airy, and its windows open day and night, irrespective of season or weather. The child should sleep alone. In addition to a warm cleansing soap bath once a week it should receive a cool sponge bath twice a day followed by brisk rubbing of the entire body. The underwear should be of thin silk or wool, and the outer garments should vary with the season of the year—always sufficient to keep the patient comfortably warm. In the absence of fever or circulatory disturbance light exercise that does not fatigue acts very beneficially. Horseback riding is highly to be recommended.

Airy room.

The value of drugs as auxiliaries in the successful management of pulmonary tuberculosis should not be underestimated. It is not very long ago that creosote was almost universally hailed as the specific against consumption. And, while its curative claims had been (as is always being done with new methods of treatment) grossly exaggerated, its efficiency to relieve distressing symptoms (useless cough), and to aid in arresting the further spread of the tuberculous lesion cannot wholly be denied. Creosote should be given in small gradually enlarged doses, well diluted in milk, malt extract or red wine. Another drug-mixture deserving of trial is the compound syrup of hypophosphites. It is a useful tonic, and may advantageously be combined with malt and cod-liver oil, as follows:—

Creosote.

Cod-liver oil.

℞ Olei morrhuæ	℥iv	120
Extracti malti,		
Syrupi hypophosph. comp.	āā ℥j	30
Glycerini	ʒiv	15
Pulveris acaciæ	ʒiv	15
Aquæ cinnamomi	q. s. ad ℥viij	240

M. Sig.: One teaspoonful three times a day.

Tonics.

The bowels should be kept open, and the appetite improved by means of bitter tonics, especially nux vomica and the tincture of cinchona compound.

Opiates.

In incipient phthisis it is very rarely necessary to resort to opiates or its derivatives to check the cough, but when the latter is distressing, especially at night, those remedies should be cautiously administered as often as indicated.

Arrest of hemorrhage.

The management of advanced cases of tuberculosis of the lungs is essentially the same as in incipient cases, except that one is often called upon to arrest hemoptysis (ice-bag to the chest, morphine hypodermatically), to check hyperidrosis (sponging of the body with a strong alum solution, atropine by mouth or hypodermatically), and to strengthen the heart's action (digitalis and strychnine). In the presence of the aforementioned complications, however, very few children survive—do what you will. Like the flickering flame of the candle end, after many ups and downs, slowly but surely, life is extinguished—often at a time when the patient seems on the mend.

TUBERCULOSIS OF THE BRAIN.

Brain tuberculosis in children occurs (1) as partial manifestation of general tuberculosis, (2) as tuberculous meningitis, and (3) as brain tumors. The brain lesions are essentially the same in the three clinical types of the disease. They consist in the deposit of tubercles in the brain substance which vary in size from a millet seed to that of a hen's egg. In tuberculous meningitis we find in addition inflammation of the pia mater of the brain and sometimes also of the cord and transudation into the ventricles (*chronic hydrocephalus*). The tubercles are usually located in the gray matter—in the large ganglia, in the pons and in the cerebellum—and occasionally also in the white substance. During life, however, it is extremely difficult to determine the seat of the lesion, except when the latter is large enough to exert pressure upon vital structures which in their turn give rise to focal symptoms—as, for example, paralysis of the cranial nerves

Variously sized tubercles.

Hydrocephalus.

in disease of the pons. In absence of such symptoms tuberculosis of the brain may exist for months without being detected. This is true especially of brain tuberculosis associated with tuberculosis of other organs. As the disease progresses, the symptomatology becomes clearer. The child suffers from intense headache, convulsions, paresis or paralysis of some of the cranial nerves or extremities, but even then it is often a matter of conjecture whether these pressure symptoms are due to tubercle or to other tumors (see Tumors of the Brain, page 524). The diagnosis is least difficult when tuberculosis of the brain is manifested by meningitis (see page 342). Here lumbar puncture often helps to clear up the diagnosis. Recourse should be had also to the tuberculin test, examination of the sputum for tubercle bacilli, and ophthalmoscopic inspection of the eyes for choroidal tubercles.

Headache, convulsions and paralysis.

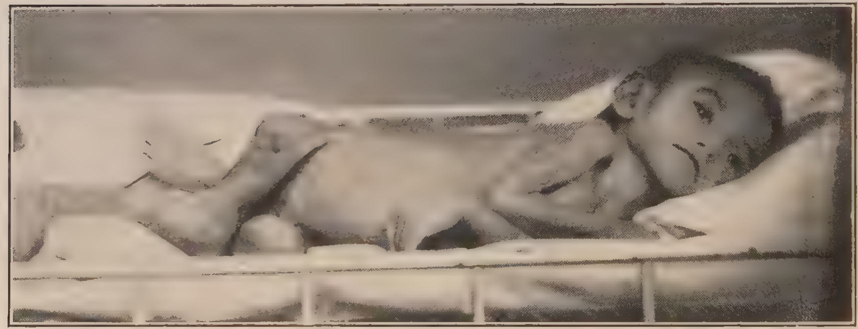


Fig. 99.—Tuberculosis of the Brain (4 years old). During the protracted course of the disease a marked hypertrichosis developed over the entire body, especially the legs. (*Sheffield.*)

TUBERCULOUS PERITONITIS.

This condition is the result of dissemination of tubercles over the peritoneum, omentum, and adjacent structures. The inflammation excited by their presence gives rise to a serofibrinous or hemorrhagic exudation with gradual agglutination of the inflamed portions, caseation and ulceration. Post-mortem examination of cases of long standing usually reveals involvement of the mesenteric and retroperitoneal glands, fatty degeneration of the liver, tuberculosis of the lungs, and parenchymatous nephritis.

Post-mortem findings.

Tuberculous peritonitis is comparatively rare in children under three years of age, but quite frequent in those over this age. The classical variety of tuberculous peritonitis is the chronic form. Occasionally, however, it may pursue a subacute, or even an acute course with chills, nausea, vomiting, acute abdominal pain, and high fever. In the majority of instances the disease sets in insidiously, with symptoms of dyspepsia, anemia, evening rise of temperature, accelerated respiration and pulse, frequent attacks of colic, and more or less pronounced diarrhea. Very soon the characteristic symptoms of the disease are in full bloom. The abdomen is distended and its wall often glistening and traversed by blue lines, the epigastric veins. The umbilicus is either effaced or protuberant. The extremities are emaciated and contrast strongly with the gradually enlarging abdomen. Palpation of the latter reveals that its consistence is

Chronic course.

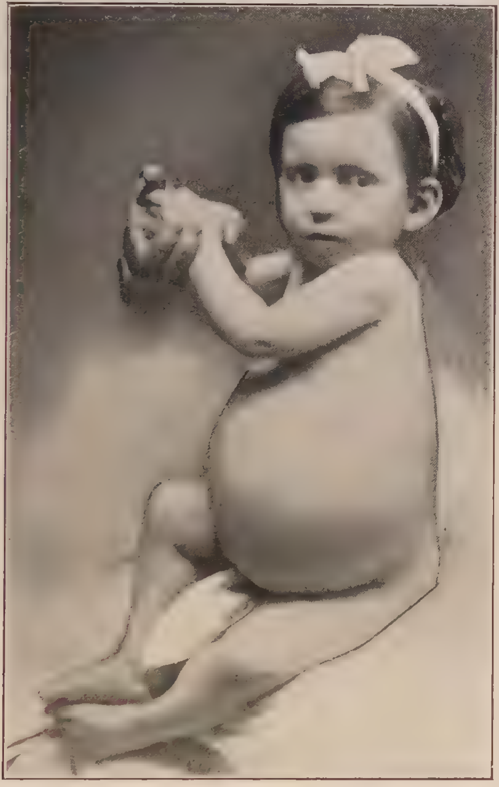


Fig. 100.—Tuberculous Peritonitis (15 months old). Recovered after Laparotomy. (*Sheffield.*)

Distended abdomen.

Emaciation.

Fluid in abdominal cavity.

not everywhere uniform. Some portions of the abdomen are flat, on percussion eliciting the presence of fluid or nodular masses; other portions again are tympanitic, denoting that that part of the abdominal enlargement is due to intestinal gases.

Cord-like masses.

Palpation sometimes confirms the findings on percussion. Occasionally hard, cord-like, painful masses and thickened omentum or adherent intestinal loops are found, and more rarely large tumors or encapsulated abscesses are detected. The latter if situated near the navel (periumbilical tuberculous abscess) may open and discharge through the navel. The abdominal enlargement may persist, or after disappearance of the fluid content and formation of fibrous adhesions the abdomen may retract, become tray-shaped, and remain so until exitus.

Hectic fever, sweats and diarrhea.

If not arrested by therapeutic measures the disease usually runs a very protracted course—months or even years. Remissions are not rare, but sooner or later the symptoms return, sometimes in acute form; the patient wastes away, is troubled by hectic fever, sweats, diarrhea, hiccough, vomiting, dysuria, anuria, and edema of the lower extremities or anasarca, until death finally relieves him of his agony. Fatal issue may occur also from intercurrent diseases, such as intestinal perforation, tuberculosis of the meninges or lungs.

On the other hand, the prognosis is not as grave if treatment is instituted early, provided, of course, that the disease is limited to the peritoneum.

Latent until late.

Unfortunately in the early stage of the disease the symptoms are not infrequently masked, and a positive diagnosis cannot be arrived at until the pathognomonic signs of the disease have made their appearance, *i.e.*, abdominal distention, circumscribed dulness, emaciation, diarrhea (diarrhea, emaciation and glandular swelling are often absent), hectic fever and swelling of the inguinal glands. Even then the peritonitis may be confounded with ascites accompanying cirrhosis of the liver or valvular heart disease. In such cases the diagnosis may sometimes be settled by the tuberculin tests, by a bacteriologic examination of aspirated adbominal fluid or by inoculation experiment.

Differentiation from hepatic cirrhosis.

Laparotomy.

As spontaneous cure is extremely rare and radical cures by laparotomy are quite frequent, the latter mode of treatment should be resorted to as soon as practicable. Some authors attribute the curative effect of laparotomy to the admission of atmospheric air to the abdominal cavity, others to hyperemia of the peritoneum produced by the operation in a manner similar to that employed by Bier in the cure of tuberculosis of the extremities. Except abundance of sunshine, sojourn at the seashore or mountains and plenty of wholesome food—which measures should be employed also in conjunction with an operation—all other medical procedures are only of temporary benefit.

Tonics.

TUBERCULOSIS OF THE ABDOMINAL ORGANS.

Aside from the intestinal tract and peritoneum, the spleen, liver, pancreas, diaphragm, omentum, suprarenals, and the urogenital system may also be the seat of tuberculous disease. Except in the rare instances of invasion of the abdominal organs by tubercle bacilli through the general circulation, the abdominal organs usually become involved secondarily to intestinal or peritoneal tuberculosis. As a rule, these latter structures become infected primarily by swallowing of food, sputum or necrotic tissue from the nasopharynx contaminated by tubercle bacilli.

Usually secondary.

INTESTINAL TUBERCULOSIS

(Tabes Mesenterica).

The tuberculous lesions are usually found in the lowest portion of the ileum, ileocecal region and colon. It is manifested by a tuberculous infiltration of the solitary follicles and mucosa of the intestine, which gradually undergo softening and caseation and finally break down, leaving behind annular ulcers. Tuberculous inflammation of the large intestine may produce so much swelling as to occlude the intestinal lumen. Sooner or later the inflammation extends to the mesenteric glands and peritoneum. Occasionally the lungs and other organs also become involved.

Caseation, ulceration and intestinal obstruction.

All these manifestations, however, are observed only at the autopsy. During life the symptoms are very obscure. Palpation may reveal enlarged mesenteric glands deep down in the abdomen, but more frequently owing to meteorism they escape observation, and even if palpable are not invariably tuberculous in nature. If, however, this symptom is associated with enlargement of other glands of the body, stubborn diarrhea (greenish-gray in color, mixed with mucus, pus, and often blood), emaciation and cachexia, sweats and hectic fever, the diagnosis of intestinal tuberculosis is fairly certain. The diagnosis is rendered positive by the demonstration of tubercle bacilli in the stools. The tuberculin test and examination of the sputa often prove decisive in

Enlarged mesenteric glands.

Stubborn diarrhea.

Tubercle bacilli in stools.

doubtful cases, and complications, such as perforation of the intestines with consecutive peritonitis, settle the diagnosis beyond doubt. Indeed, in the majority of instances the diagnosis cannot be made until these complications arise, a period at which therapeutic measures almost invariably fail. At all events the prognosis is extremely grave.

Cases of local tuberculosis detected early and treated energetically (chiefly surgically) may recover.

TUBERCULOSIS OF THE GENITOURINARY TRACT.

Urogenital tuberculosis, especially tuberculosis of the kidneys, is quite common in children. It occurs either as a manifestation of general tuberculosis or as an independent disease. In the latter event it almost invariably begins in one kidney, and from here it spreads to the bladder and the other kidney. In the beginning the affection is very apt to be overlooked, but, as the tuberculous process advances, the symptoms (pain in the region of the kidney and ureter, thickening of the ureter—as evinced by palpation with the finger in the rectum or vagina—irritability of the bladder, albuminuria, pyuria, and often hematuria) become sufficiently characteristic as to demand careful, repeated, bacteriologic examination of the urine for tubercle bacilli, and cystoscopic inspection of the bladder for tuberculous lesions. Even in the early stage systematic cystoscopic examination of the bladder will rarely fail to detect tuberculous nodules and ulceration about the opening of one ureter (see Fig. 103). In cases of long standing the lesions are often found scattered throughout the bladder. As in tuberculosis of other organs the tuberculin test should always be employed to corroborate the diagnosis. Early recognition of the condition and prompt surgical treatment are not rarely followed by permanent recovery.

Pyuria, hematuria and tubercle bacilli in urine.

SCROFULOSIS

(Tuberculosis of the Skin, Mucous Membranes and Glands).

The tuberculous nature of the symptom-complex embraced by the term "scrofula" is no longer a matter of dispute. The disease attacks children with undermined constitution who are poorly fed and cared for, are forced to live in damp, dark and filthy dwellings, and are exposed to tuberculous infection. Various skin eruptions, or injuries, exanthemata, decayed teeth, and diseased tonsils and adenoids, among others, serve as the

Portals of entry.

portals of entry to the tubercle bacilli. The immediate result of the tubercular infection is hyperplasia, and the more remote effect, caseous degeneration of the parts primarily involved, and frequently secondary infection of the neighboring structures.

Hyperplasia and caseous degeneration.

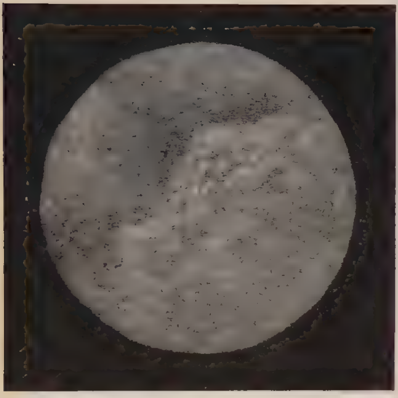


Fig. 101.—A characteristic early tubercular infiltration, as seen through the cystoscope. (*Leedham-Green.*)

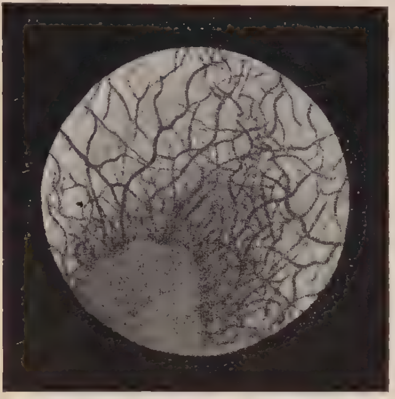


Fig. 102.—A large tubercular ulcer below the orifice of the right ureter. (*Leedham-Green.*)

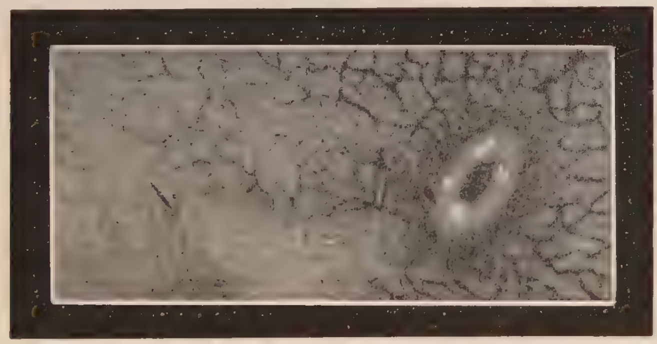


Fig. 103.—Cystoscopic view of the base of the bladder in a case of tuberculosis of the left kidney (*Wyatt*). The opening of the right ureter is normal; the opening of the left ureter is seen to be gaping, the lips edematous and thickened, showing the presence of small miliary tubercles.

Clinically scrofulosis is characterized by simultaneous or successive involvement of the skin, mucous membranes and lymphatic glands; chronicity of its course, and a tendency toward slow spontaneous recovery, or transition into general tuberculosis.

Chronic course.

Suppuration. Ulceration.

The skin is the seat of a pustular eruption which resists ordinary local treatment, generally involves the subcutaneous tissue, and breaks down, forming slowly discharging abscesses or indolent ulcers. It is most frequently situated upon the back and nates, but is found also upon the scalp and face—probably carried from one part to the other by scratching by means of infected fingers.

Scrofulosis of the mucous membranes is manifested chiefly by nasopharyngitis. From the nasopharynx the inflammatory process may spread to the ears, eyes, larynx and oral cavity.

Naso-pharyngitis. Otorrhea. Chondritis and periostitis.

The nasal mucous membrane is red and swollen and discharges a seropurulent secretion which forms yellowish-green crusts within and around the nares, producing snuffling respiration, and excoriation of the upper lip. A similar acrid discharge is usually observed from the ears (bilateral otorrhea). Both the nasal and aural discharges may become purulent and fetid, in the first instance, by extension of the inflammation from the nasal mucous membrane to the cartilage, periosteum and even nasal bones (sometimes marked nasal deformity); in the second instance, by implication of the middle ear and eventually the ossicles, or petrous portions of the temporal bones.

Phlyctenular keratitis.

Scrofulosis of the eyes, the so-called *strumous ophthalmia,* usually begins with redness and swelling of the palpebral mucous membrane, and in the majority of cases is soon followed by involvement of the cornea, in the form of *phlyctenular keratitis,* with strong lacrimation, pain, and photophobia. The phlyctenulæ are very slow in healing, and show a great tendency to leave behind corneal opacities. Blepharoadenitis, madarosis and permanent thickening of the edges of the lids are quite common accompaniments.

Fistulæ.

The lymphatic glands are affected early or late—secondarily to the inflammation of the skin and mucous membranes. Except their wide distribution the glandular swellings present nothing characteristic in the beginning, but as the disease progresses they show a marked tendency to undergo caseation and suppuration. Furthermore, after evacuation of the pus which usually contains tubercle bacilli they rarely cicatrize, but, on the contrary, continue as pus-discharging fistulæ or indolent ulcers.

The course of the disease depends greatly upon the vitality of the patient and the mode of treatment. It is always chronic. Children removed from the obnoxious surroundings frequently recover completely. In those not properly cared for the tuberculous process is very prone to spread to the osseous system and to the internal organs. Spina ventosa, osteomyelitis and spondylitis form frequent sequelæ (for details of these affections the reader is referred to the chapter on "Tuberculosis of the Bones," page 374). The internal organs, especially the liver, spleen and lungs, may be implicated singly or collectively, in which event the prognosis, of course, is extremely bad.

Spina ventosa.

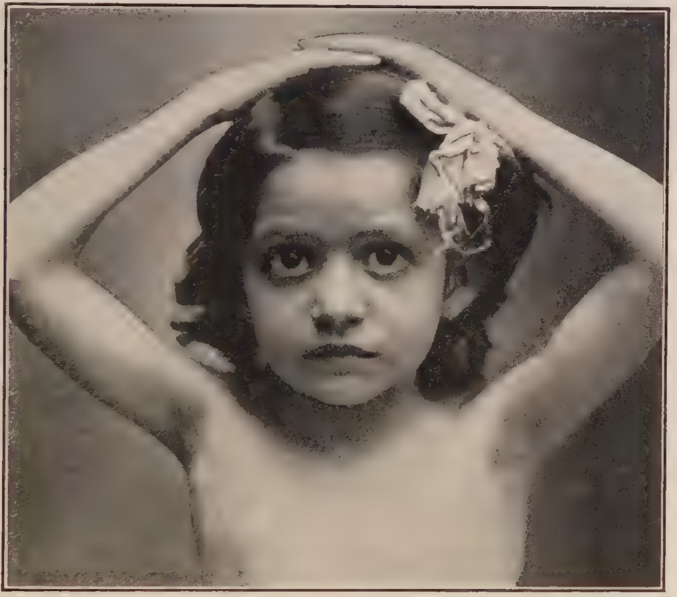


Fig. 104.—Tuberculous Axillary Lymphadenitis. (*Sheffield.*)

Characteristic as the symptom-complex of scrofulosis seems to be, errors of diagnosis are nevertheless very apt to be made. The perplexity is often great in the differentiation between scrofula and inherited syphilis, both of which diseases have many symptoms in common. In all such doubtful cases it is wise, on the one hand, to employ the tuberculin reaction, and examine the aural and nasal secretions as well as the pus from scrofulous abscesses for tubercle bacilli, and, on the other, to administer mercury and look for the spirochæte pallida. One should not be too hasty in pronouncing a case as scrofulosis because of the so-called "torpid habitus" of the patient (pale, flabby, puffed face; thick nose, swollen and excoriated upper lip, redness and thick-

Resembles syphilis.

Tubercle bacilli in scrofulosis; spirochætes in syphilis.

ening of the lids), or the presence of adenoids or glandular swelling. These symptoms can and often do exist independently of tuberculosis.

Prompt and energetic treatment.

Scrofula, like other forms of tuberculosis, demands early and energetic treatment. The patient should be removed from the obnoxious influences, well nourished and kept outdoors the greater part of the day (see page 352). Internally we should administer, for several months in succession, moderately large doses of the syrup of the iodid of iron and the syrup of hypophosphites, as well as cod-liver oil or similar alterative tonics.

Tonics.

Bodily cleanliness.

The local treatment, which is of very great importance, essentially consists of thorough bodily cleanliness (daily bath with sea salt; antiseptic dressings to open wounds, etc.); removal of diseased foci (*e.g.*, tonsils and adenoids, decayed teeth, caseated glands, etc.), and evacuation of pus wherever found. Individual complications should be vigorously combated according to indications. (See bone tuberculosis, below; otitis, page 250; eczema, page 591, etc.) As the external lesions are probably the result of carrying infectious material from place to place by means of the fingers, open wounds (vaccination wounds!) should be thoroughly protected and the patient's finger-nails clipped and kept scrupulously clean to prevent scratching the diseased parts of the body and direct infection of its healthy portions.

Protection of open wounds.

℞ Syr. ferri iodidi ....ʒiij | 12
Syr. hypophosph. co. ....q. s. ad ℥ij | 60
M. Sig.: ʒj three times a day for a child 3 years old.

oteo - infla. of
arrow of bone.

TUBERCULOSIS OF BONES AND JOINTS
(Tubercular Osteomyelitis and Arthritis).

arthritis - infl
joint.

The grouping together of tuberculous bone and joint diseases is intended to emphasize their correlation. The favorite seat of bone tuberculosis is usually in the epiphyses, the joint becoming involved secondarily by extension of the inflammatory process to the synovial structures. Occasionally the joint is affected primarily.

Primary or secondary.

The immediate cause of the disease is the tubercle bacillus which invades the medullary tissue, the bone proper, or the articular structures, either from within—from a florid or latent tuberculous focus elsewhere—or from without—as a result of traumatism. An inherited predisposition and impaired nutrition from various causes favor the development of tuberculous disease.

Osseous as well as articular tuberculosis is essentially a chronic inflammatory process, free from the violent symptoms which are characteristic of acute, non-tuberculous osteomyelitis. Extensive lesions may exist for weeks and months with apparent perfect health. Fever is usually absent in the beginning and only slight—in the evening—at a later stage of the disease. As the tuberculous process advances progressive anemia and emaciation make their appearance but are not pathognomonic of the affection. The local symptoms also are very vague at first. Hence the reason why local tuberculous disease is frequently

Chronic process.

Emaciation.

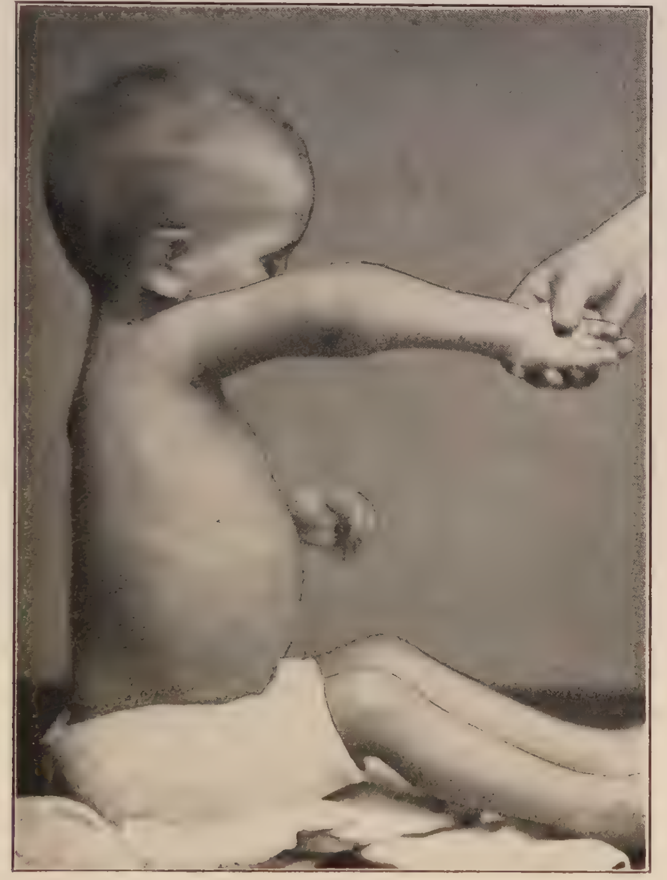


Fig. 105.—Tuberculous Disease of the Elbow-joint in Boy 18 Months Old. (*Sheffield.*)

Frequently overlooked.

overlooked until, as will presently be shown, deformity and loss of function have occurred, which vary greatly in extent and severity with the seat of the lesions and the mode of treatment.

1. TUBERCULOSIS OF THE VERTEBRAL COLUMN

(Spondylitis; Pott's Disease).

The tuberculous process usually begins in or near the vertebral body, and if not arrested, gradually extends to the contiguous structures, including the spinal cord.

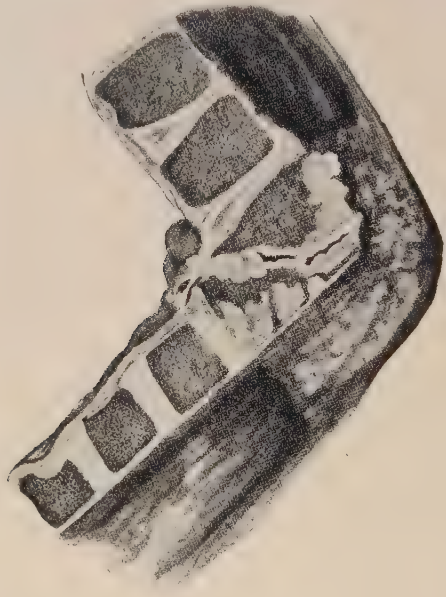


Fig. 106.—Pott's Disease (*Langerhans*). Kyphosis of dorsal vertebræ, the result of caseous tuberculous periostitis and osteomyelitis. Destruction of three thoracic vertebræ. Two-thirds natural size.

It is manifested by an ulcerative and often suppurative destruction of the bone, with metastatic—gravitation—abscesses in distant locations, *e.g.*, retropharyngeal abscess, in cervical spondylitis; psoas abscess, in lower dorsal and lumbar disease. Furthermore, with softening and crumbling of the vertebral bodies, the spinal column, as it were, topples over, usually backward, producing a deformity known as kyphosis, gibbus or Pott's hump. The condition is gradually further aggravated by compensatory spinal deformities (especially lordosis) and a group

Softening and crumbling of vertebral bodies.

Kyphosis.

Lordosis.

of other distressing pressure symptoms soon to be related which if not arrested throw the unfortunate creature in an abyss of everlasting misery.

Slow onset and course. This process, fortunately, is very slow in development, affording ample time—from three to ten years—to arrest and mend its ravages and ample warnings to the patient to seek relief. **Four stages.** We may frequently differentiate four stages in the progress of the affection: 1. The stage of onset, where the symptoms are very vague and inconstant. The child shows a disinclination to play, refuses to walk or tires easily when it does walk. It complains of pain in different parts of the body, following the distribution of the spinal nerves, **Pain, especially at night.** the pain being often so severe, especially at night, that it wakes the child from its sleep with a sudden

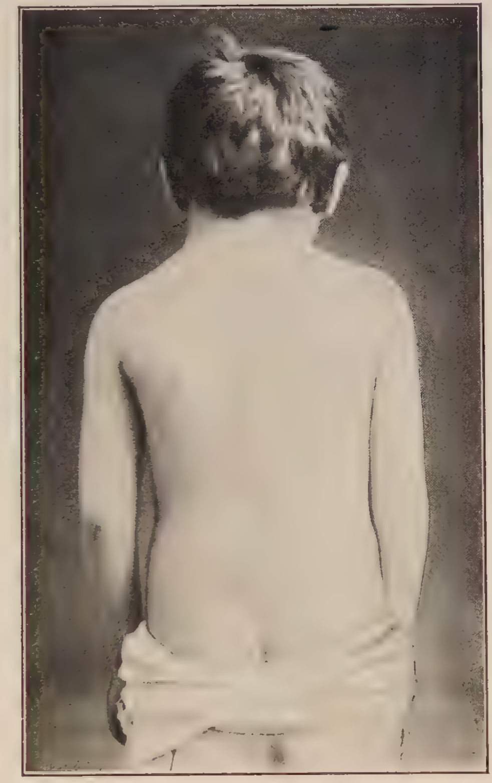


Fig. 107.—Cervical Spondylitis. Note broadness and tilting of neck. (*Sheffield.*)

start—"starting pain." 2. The stage of fixation of the spinal column; 3, the stage of characteristic deformity; and 4, the stage of suppuration and pressure paralysis. The disease does not always progress to the last stages. In some instances, after two or three years' course, either through treatment or spontaneously,

Starting pain.

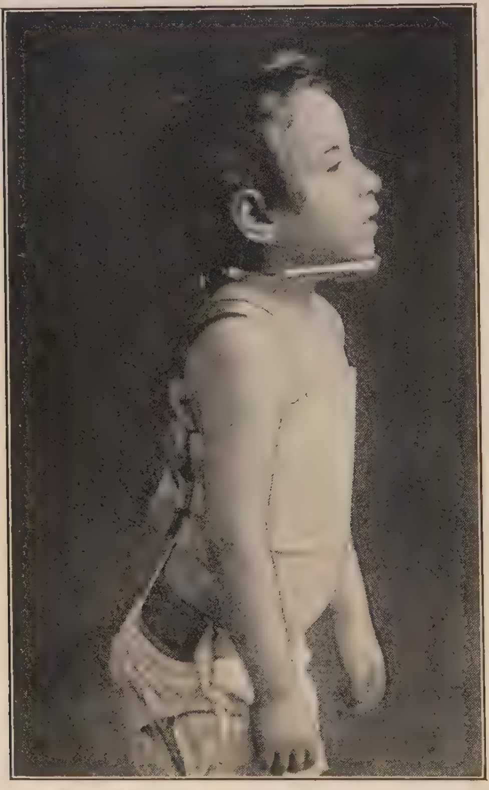


Fig. 108.—Cervical Spondylitis. Same case as Fig. 107, in brace. (*Sheffield.*)

solidification of the diseased vertebræ and relative cure occur. Relapses, however, are not infrequent. Pressure paralysis (see Myelitis) is especially common in disease of the lower cervical and upper dorsal, and rare in that below this region.

Pressure paralysis.

The focal symptoms vary with the seat and extent of the lesion. In cervical spondylitis the patient, if old enough, complains of neuralgic pain in the head and upper portion of the neck. Very young children indicate the presence of pain by suffering

Cervical spondylitis.

and anxious expression of the face, by refusal of food and crying on handling. The head is stiff, tipped backward, or laterally (torticollis-like), and when the child moves it is often seen to support its head with the hands. At a later stage of the disease, there are often disturbances of deglutition and voice—not rarely due to retropharyngeal abscess. If the uppermost cervical vertebræ are diseased, there is danger of anterior displacement of the head between the atlas and axis, more rarely between the occiput and atlas, and death from pressure upon the cord. The permanent deformity in cervical spondylitis usually consists of thickening and broadening of the neck, and sinking of the head upon the shoulders.

Torticollis.

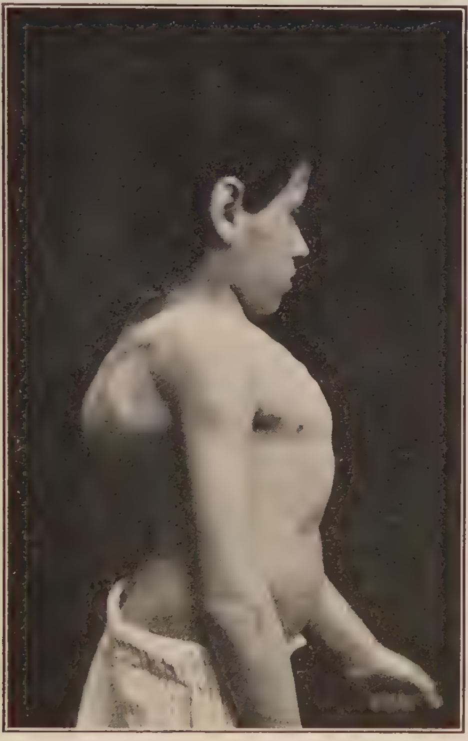


Fig. 109.—Dorsal Spondylitis, Gibbus (12 years old). (*Sheffield.*)

Dorsal spondylitis.

In dorsal spondylitis the distribution of the pain differs somewhat with the particular part of the spine involved. If the *upper* dorsal vertebræ are affected, the pain resembles that of intercostal neuralgia, and increases on coughing, sneezing, laughing, etc., while in spondylitis of the *lower* dorsal vertebræ, the most frequent seat of the disease, the pain radiates to the lower extremities. In disease of this region, furthermore, the upper part of the body deviates to the side, one shoulder is elevated and the trunk bent to the opposite side—a state of scoliosis; at

Scoliosis.

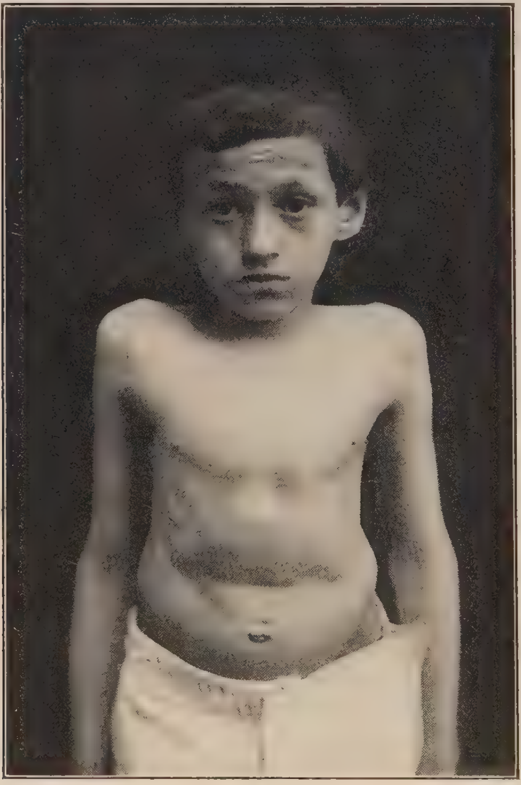


Fig. 110.—Dorsal Spondylitis. Same case as Fig. 109, front view. (*Sheffield.*)

the same time the vertebral column is kept rigid, every movement carefully avoided, and in walking short rigid steps are taken, the patient timidly balancing the superincumbent weight of the body by firmly supporting the spine with the hands. If urged to pick up something from the floor, the child stoops by strongly flexing the knee- and hip-joints, while holding the vertebral column perfectly rigid, and raises himself by resting the hands upon the thighs, and then, with alternating supporting

Characteristic attitude in walking and stooping.

movements along the thighs and trunk, elevates the body and lastly extends the legs. If bending of the spinal column is attempted, motion occurs only in the healthy sections, the diseased portions remaining firmly fixed. The ultimate spinal deformity consists of kyphosis, kyphoscoliosis and lordosis.

Attitude in bending.

Kyphoscoliosis.

In lumbar disease the patient complains of pain in sitting, and refers it also to the lowest portion of the abdomen and the legs. The physical signs are essentially the same as in spondylitis of the lower dorsals, except that the deformity occurs at a later period and is not as pronounced. On the other hand, there is a greater tendency toward the formation of psoas abscess—a tumor deep in the iliac fossa or at the anterior surface of the thigh, lameness and flexion of one thigh.

Psoas abscess.

Careful attention to the aforementioned physical signs rarely fails to disclose the presence of vertebral caries, even at an early stage of the disease. Cervical spondylitis may be mistaken for torticollis (sudden onset, pain and unilateral contracture more pronounced; early response to anodynes and antirheumatics, etc.); for cervical rib (revealed by X-rays); non-tuberculous retropharyngeal abscess (immediate relief on puncture). Dorsal and lumbar spondylitis may be confounded with rachitic curvature (rounded in rickets; angular in spondylitis; rachitic kyphosis is reducible by placing the child upon the abdomen and over-extending the thighs; absence of characteristic gait and mode of stooping). Right iliac psoas abscess often resembles appendicitis (onset sudden or recurrent, rigidity of the abdominal muscles, absence of spinal disease). Psoas abscess differs from hip-joint disease by the hip-joint being fixed in the latter affection; and from hernia by being reducible in recumbent posture.

Differentiation from torticollis, cervical rib, retropharyngeal abscess and rachitic kyphosis.

Differentiation of right psoas abscess from appendicitis, coxitis and inguinal hernia.

In view of the comparatively slow course of the disease in the majority of cases, the prognosis as to life is good, and as to permanent deformity fair, *provided* the treatment is begun early and persisted in. The prognosis is bad in cases presenting abscesses, fistulæ, and pressure paralysis. Even here surprisingly good results are often obtained under suitable treatment.

The treatment is principally orthopedic and surgical—fixation of the spine by a plaster of Paris or (in milder cases) celluloid jacket, rest in bed to unburden the spinal column, and evacuation of large collections of pus (*e.g.,* retropharyngeal or psoas abscesses). Good hygiene, outdoor air, plenty of nutritious food, and iron, hypophosphites, and cod-liver oil will facilitate a cure.

Fixation.

Evacuation of pus.

SCOLIOSIS[1]

(Lateral Curvature of the Spine).

Habitual.

In contrast to the aforementioned spinal deformities, this form of scoliosis is not tuberculous. As a rule, it is habitual in nature, the result of unequal (one-side) compression of the intervertebral cartilages, favored by atony of the muscles and ligaments and weakness of the bones. It is most frequently observed in school children, especially girls, and is generally ascribed to faulty posture while sitting at the school desk, etc., and to the habitual carrying of heavy books with one arm. I firmly believe that a great many cases of the so-called habitual

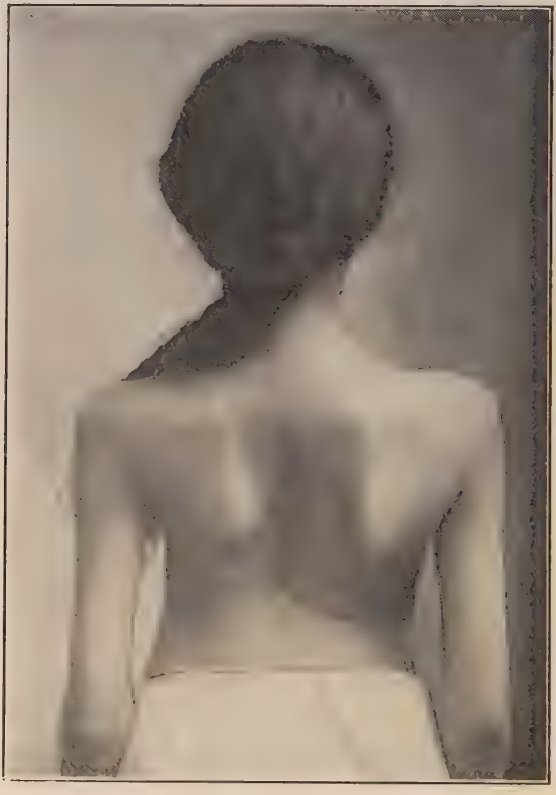


Fig. 111.—Lateral Spinal Curvature. Second degree. (*Sheffield.*)

Unequal compression of inter-vertebral cartilages.

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[1] This spinal, non-tuberculous deformity is discussed here in order to emphasize its differences from spondylitis.

Rachitic.

lateral spinal curvatures originate during early infancy in connection with rachitis (*q. v.*), are generally overlooked while the deformity is slight and are detected later, at a time when the deformity does and would gradually get worse, whether or not the child goes to school. Of course, this view does not preclude the fact that faulty posture and encumbrance of one-half of the body hasten to aggravate the curvature.

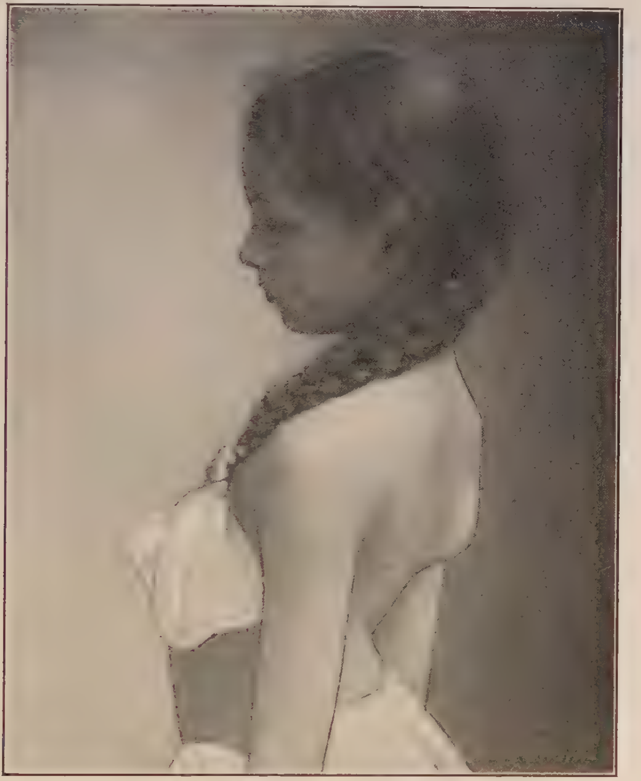
Fig. 112.—Lateral Spinal Curvature. Same case as Fig. 111. Side view. (*Sheffield.*)

Static.

Paralytic.

Cicatricial.

Less frequent causes are obliquity of the pelvis (*e.g.*, shortening of one lower extremity from birth or postnatal disease); unilateral paralysis (*e.g.*, poliomyelitis, progressive muscular atrophy); unilateral immobility of the thorax (*e.g.*, protracted extensive pleuritic exudation or adhesions); and unilateral sinking of the thorax from traumatism or operations (*e.g.*, multiple fractures of ribs, resection of ribs in pyothorax). Very rarely scoliosis is congenital in nature, when, as a rule, it is associated with other congenital malformations.

Congenital.

High shoulder.

Scoliosis is manifested first by elevation of one shoulder, and later by prominence of one hip and scapula on the same side and gradually increasing convexity of the spinal column and side. With further progress of the deformity, the spinal column presents two curves, in the shape of the letter S (see Figs. 115 and 116)—the primary curve, which is usually in the dorsal region, and the secondary or compensatory curve, usually in the lumbar region. Bad cases are occasionally complicated also by lordosis, deformity of the thorax and displacement of the heart and lungs, but are otherwise free from constitutional symptoms.

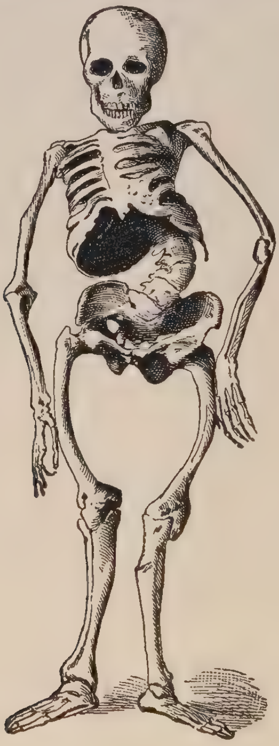

Fig. 113.—Rachitic scoliotic skeleton. (*Grandin, Jarman* and *Marx.*)

S-shaped curvature.

Lordosis.

Fortunately, nowadays, with the greater attention being paid to the general health of children, these dreadful deformities are very rarely encountered.) Many cases come under the care of the physician in the first stage of the disease which ordinarily yields to massage, calisthenics, fresh air, ample nutrition, general medicinal tonics, and, above all, removal of etiologic factors. (Severer forms of scoliosis are often corrected by a plaster-of-Paris or celluloid corset—worn continuously for several months, and followed by massage and exercise to strengthen the weak muscles. (Fixed scoliosis can at best only be impeded in its further progress, but the damage done is frequently irreparable. Hence, the importance of early and energetic treatment, and particularly of prophylactic measures, which are especially effective in habitual scoliosis. Here the school physician is offered many opportunities to merit the gratitude of the community.)

Calisthenics.

Plaster-jacket.

Prophylaxis.

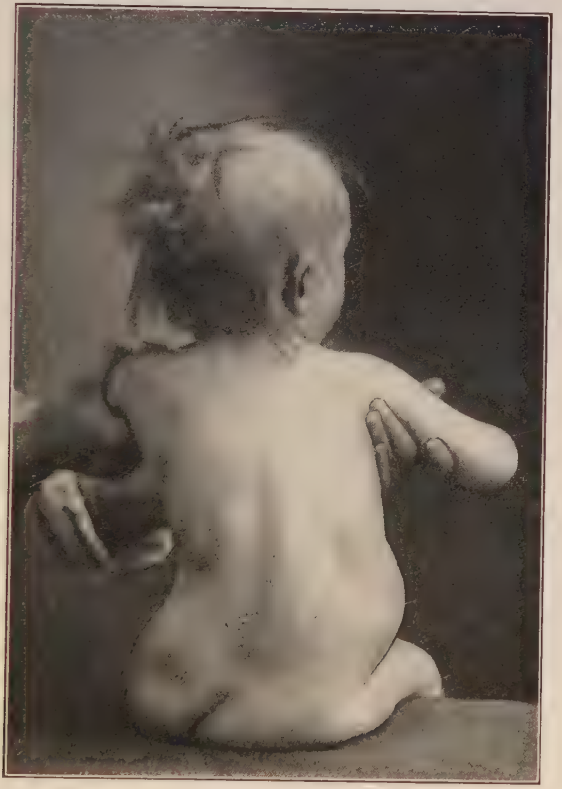

Fig. 114.—Paralytic Scoliosis. Same case as Fig. 173, posterior view. (*Sheffield.*)

2. MORBUS COXARIUS

(Hip-joint Disease, Coxitis Tuberculosa, Articular Osteitis of the Hip).

Osteitis.

Suppuration.

Displacement.

The pathologic process of this tuberculous affection is usually described as consisting of three stages: 1, the stage of osteitis, as a rule, involving the femoral head, less frequently the acetabulum; 2, the stage of arthritis or suppuration, in which all the joint structures are implicated; and 3, the stage of disintegration and absorption of the head and sometimes the neck of the femur and the upper and back part of the acetabulum, with "wandering" of the head of the femur upward and backward upon the dorsum ilii.

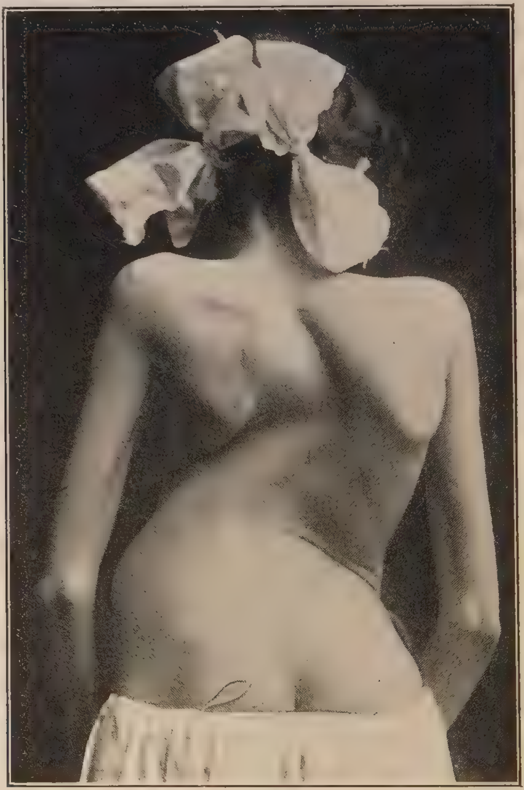

Fig. 115.—Lateral Spinal Curvature (S-shaped scoliosis; see page 384). (*Sheffield.*)

Simultaneously with the onset of the first stage of the patho-

Limp.

Pain in knee.

logic process, or sometimes at a later period, the child begins to limp and to complain of pain in the knee- or hip-joint or both. As a rule, the limp at first is intermittent in character, more marked either in the morning or in the evening, but as the inflammation progresses it becomes constant and quite pronounced, the leg at the same time being held very rigid. With the occurrence of articular exudation, the leg assumes a pathognomonic position of flexion, abduction and eversion, and the patient in order to bring the foot to the ground depresses the pelvis on the affected side, thus giving rise to slight—apparent—lengthening of the limb. With destruction of the joint and the articular bony structures, the hip-joint becomes further flexed, inverted and adducted. To overcome the uselessness of the limb in this position the

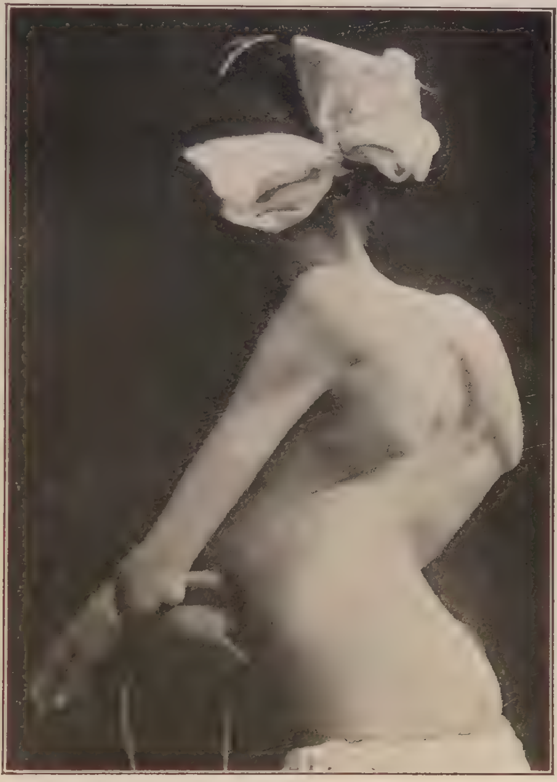

Fig. 116.—Lateral Spinal Curvature (S-shaped scoliosis). Same as case Fig. 115. Side view. (*Sheffield.*)

Rigidity.

False position of leg.

Apparent lengthening.

Apparent shortening.

patient elevates the pelvis on the affected side, and to counteract the—apparent—shortening he steps on the ball of the foot. Later real shortening ensues, owing to the wandering of the femoral head upward and backward, and firm contraction and atrophy of the muscles.

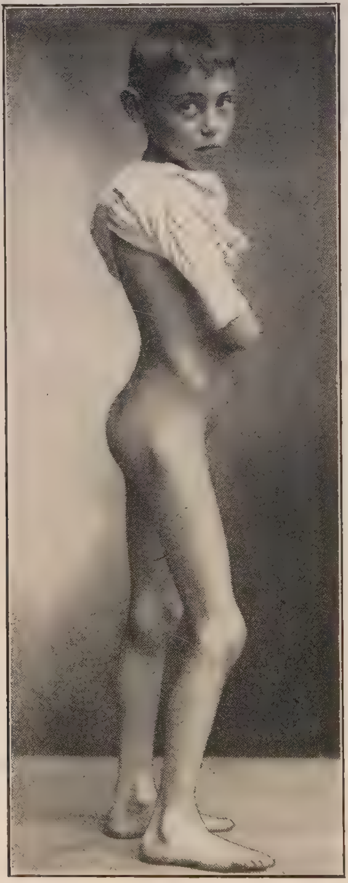

Fig. 117.—Hip-joint Disease. (*Sheffield.*)

Compensatory scoliosis and lordosis.

In upright posture, in consequence of the pelvic obliquity, the patient assumes a position of compensatory scoliosis and lordosis. In recumbent posture, with the limbs brought down parallel to each other, there is always compensatory lordosis of the lumbar region. This lordosis disappears on flexing the affected limb at the hip to an angle at which it is held flexed by the contracted muscles.

Starting pain.

The intensity of the pain varies. It is usually worse after manipulation and fatigue, and at night. It may awaken the child from its sound sleep with a cry (*"starting pain"*). The pain not rarely is referred to the knee, or to other parts supplied by the obturator nerve, *e.g.,* the inner side of the thigh. Hence the importance of always examining the hip-joint in such cases.

Tumefaction.

In addition to the pain, the limp and false position, we may find at a late stage of the disease involvement of the inguinal glands, with or without suppuration and perforation; enlargement—"white swelling"—of the hip; flattening of the gluteal region and effacement of one gluteal fold; multiple abscesses and fistulæ at various points of the hip or thigh, especially at the

Irregular temperature.

tensor fasciæ latæ; irregular temperature, especially during the stage of suppuration.

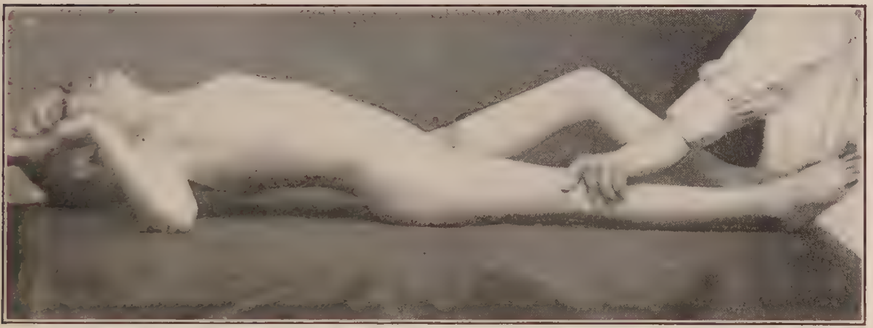

Fig. 118.—Hip-joint Disease. Note compensatory lordosis on full extension of affected limb. (*Sheffield.*)

Exhaustion.

Cases presenting the aforementioned typical symptoms are recognizable at a glance. Indeed, at this late stage of the disease, it is almost immaterial whether a correct diagnosis is made or not, since a fatal issue from exhaustion, amyloid degeneration and general tuberculosis is all that can be expected, particularly in children with a tuberculous diathesis. The center of the physician's interest therefore should rest upon the diagnosis of incipient coxitis which, if properly treated, offers good prospect of recovery. A history of slight trauma; occasional dragging of the leg or limping; pain in the hip- or knee-joint; disinclination to play and undue fatigue after slight exertion; restless sleep and "starting pain," all point to coxitis and demand very careful and repeated examinations of the hip-joint. The diagnosis is greatly facilitated and in the majority of instances rendered positive by the presence of pain on pressure against the trochanter, or against the acetabulum (by digital rectal examination), and von Pirquet's tuberculin test. Advanced coxitis can readily be diagnosed by the aforementioned faulty attitude of the patient, in recumbency, standing, or walking. In doubtful cases, an X-ray examination (by an experienced radiographer) is decisive. The latter procedure is especially useful in differentiating coxitis from: Injury to the hip (disability follows immediately after the accident; local signs of injury, *e.g.*, ecchymosis, etc.); coxa vara (X-ray shows downward inflexion of the neck of the femur; adduction and extension of the limb are usually

X-ray in differentiation from trauma of hip and coxa vara.

Differentiation from Congenital dislocation of hip; osteomyelitis; rheumatism; lumbar spondylitis; hysteria,

possible); congenital dislocation of the hip (history of lameness from birth; absence of inflammatory signs or limitation of motion); osteomyelitis with separation of the epiphyses (very violent course); rheumatism (yields to the salicylates; no bone lesion); spondylitis of the lumbar region (distinct symptoms of spondylitis; hip-joint free); hysteria (absence of joint trouble, best proven under anesthesia, and by means of X-rays); periosteal sarcoma of the trochanter (see Fig. 119) (swelling rapidly increases in size; marked dilatation of the superficial veins).

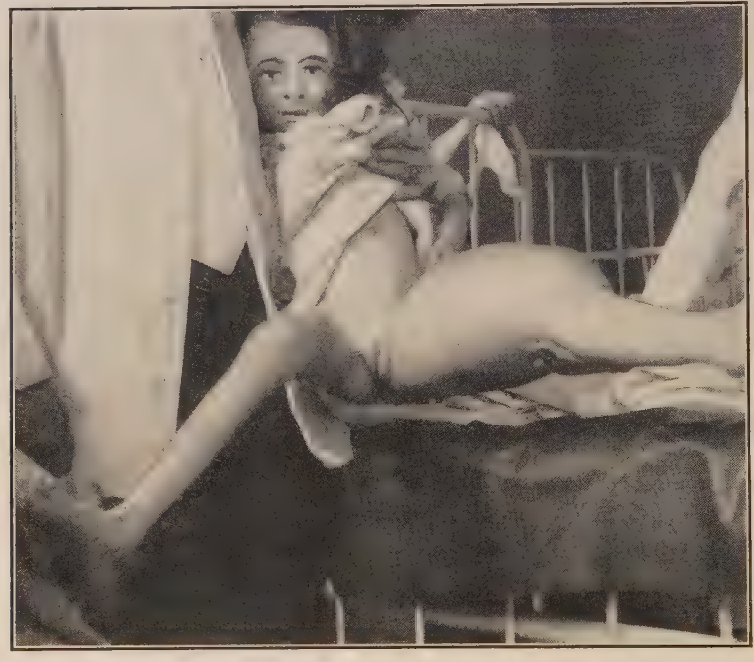

Fig. 119.—Sarcoma of the Femur in a Girl 8 Years Old. (*Sheffield.*)

and sarcoma of upper part of femur.

Reduction of deformity, fixation and rest.

The treatment consists of: reduction of existing deformity, either gradually (by weight and pulley, while the patient is in bed) or forcibly (under anesthesia); disencumbrance of the hip-joint of the body weight, at first by rest in bed (bed extension apparatus, so as to enable the patient to enjoy fresh air) and later by means of an extension-walking apparatus; and, finally, fixation of the hip-joint by a plaster-of-Paris spica or a fixation apparatus. Fixation of the joint as well as extension should be continued for some time after apparent recovery. Constitutional treatment. Massage to prevent atrophy of the muscles and stiffness of the healthy joints.

Massage.

3. KNEE-JOINT DISEASE

(Tuberculosis of the Knee-joint. White Swelling).

The pathologic process of tuberculosis of the knee-joint resembles that of the hip. It may begin in the synovial membrane or in the articular ends of the osseous structures. The clinical symptoms are practically the same, whether the synovialis has been affected primarily or secondarily. They consist of fusiform swelling, local tenderness, atrophy of the thigh and calf

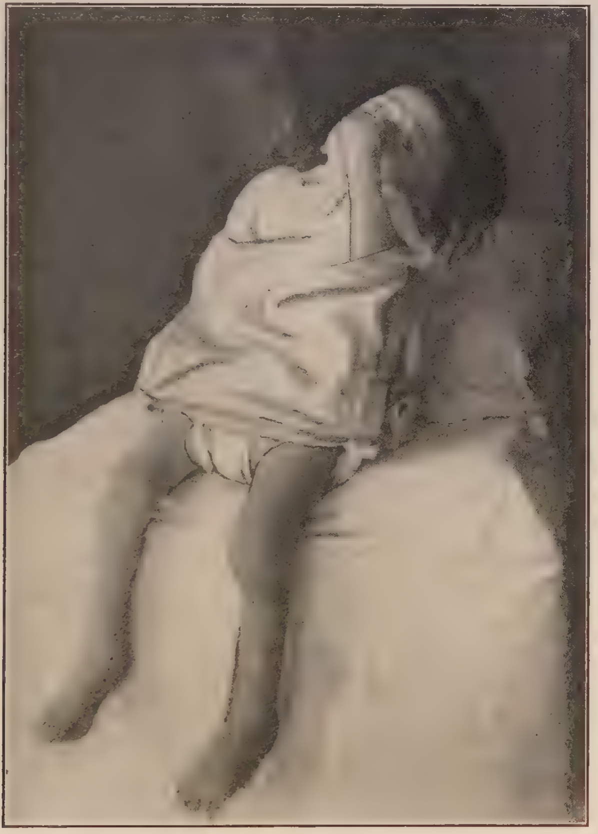

Fig. 120.—Tuberculous Disease of the Knee-joint in a Child 13 Months Old. Patient succumbed to tuberculous pyothorax. (*Sheffield.*)

Fusiform swelling.

muscles, flexion and slight outward rotation of the knee, and later abscess formation (extra- or intra-articular). During the suppurative stage, less frequently in the absence of suppuration, there are more or less constitutional symptoms, such as anorexia, anemia, emaciation and irregular fever. The latter is quite high in the presence of secondary infection.

Constitutional symptoms.

The tuberculous process pursues a rather slow course. Not rarely it is interrupted by prolonged remissions. Exacerbations are often induced by local trauma or intercurrent acute diseases, sometimes after an "apparent" cure had been established. The prognosis as a whole, however, is favorable, if treatment is begun early and properly. The very rarely occurring spontaneous recovery should not be depended upon.

Remissions.

Within recent years the treatment of tuberculosis of the knee-joint, as well as that of the other smaller joints, has been entirely revolutionized. Instead of resorting to immobilization, resection and permanent fixation, Bier's method of passive hyperemia has become the treatment of choice, since it not only aids nature in the healing of the tuberculous process, but tends also to restore the normal functions of the affected joint. The mode of procedure is very simple. A soft-rubber bandage about 2 inches in width is applied gently and evenly around the extremity, at some distance above the lesion, *e.g.*, at the middle or upper third of the femur in tuberculosis of the knee-joint, and left in place for an hour or two, once or twice a day. If the bandage is properly applied it gives rise to no pain, nor interruption of the pulse. The extremity below the bandage soon swells slightly, and assumes a bluish-red color, but remains warm. The favorable results obtained from this mode of treatment of tuberculous joints are rather slow in coming (from three to nine months), but in uncomplicated cases well worth waiting for. Complications arising should be treated symptomatically. Thus cold abscesses call for free incisions and evacuation (may be enhanced by cupping-glass) of the necrosed tissue; large exudations should be treated by aspiration and injection of iodoform emulsion, and the general health should be improved by outdoor fresh air, nutritious food, tonics (iron and cod-liver oil), massage and hydrotherapy. For differential diagnosis, see "Arthritis," page 419.

Bier's passive hyperemia.

Surgical treatment.

4. SPINA VENTOSA

(Tuberculosis of the Metacarpals and Phalanges. Tuberculous Dactylitis).

Usually first phalanx.

This disease most frequently affects the first phalanx of the index finger, but may occasionally be found simultaneously in several phalanges or metacarpals of the same hand. The osseous tissue is gradually destroyed, and, while this is going on, here and there new bone tissue is gradually formed under the periosteum. In consequence of the latter process, the finger becomes fusiform, as if the bone had been "blown up" (see Fig. 121). As the inflammatory process is very slow and painless, it, as a rule, takes several months before the characteristic appearance is developed. At a later stage of the disease, there is circumscribed redness, fluctuation, impairment of function of the tendons and spontaneous rupture of the suppurating focus with very tedious discharge of the contents.

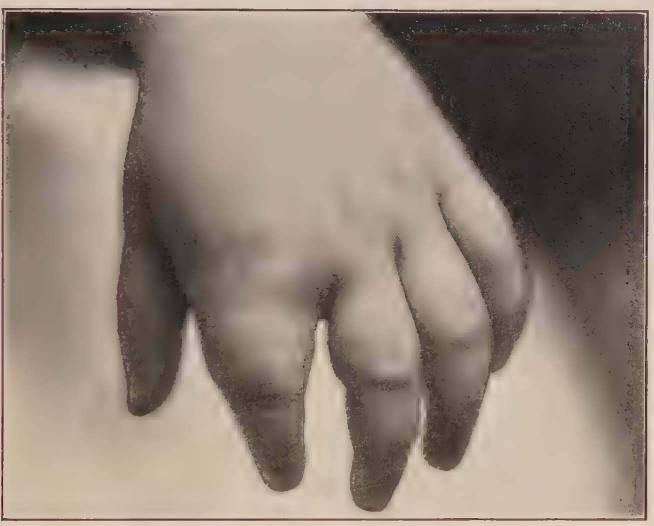

Fig. 121.—Spina Ventosa. (*Sheffield.*)

Fusiform swelling.

Differentiation from syphilis.

Tuberculous dactylitis may be mistaken for a congenital or acquired syphilitic lesion. The history of syphilis, the presence of other syphilitic symptoms, the greater tendency of syphilitic dactylitis to be multiple and symmetrical, and the ready response to antisyphilitic treatment usually suffice to clear up the diagnosis. A positive von Pirquet tuberculin test and the coincidence of tuberculous lesions elsewhere point strongly to tuberculosis.

Conservative surgery.

Early constitutional treatment and passive hyperemia (see page 392) are very efficient curative measures. Conservative surgery (evacuation of pus and sequestra) is indicated in neglected cases. In these recovery is slow, usually with permanent deformity.

NON-TUBERCULOUS OSTEOMYELITIS

(Osteitis; Periostitis).

The term osteomyelitis refers chiefly to inflammation of the marrow of the bone, but includes also the morbidity of the bony matrix and periosteum, which at one period or another participate in the destructive processes.

Affects long bones.

Microbic infection.

Osteomyelitis is exceedingly common in children below the age of puberty—before completion of ossification of the epiphyses and diaphyses—since the anatomic peculiarities of the circulation in growing bones particularly favor its development on slight provocation. The affection is observed in two forms: Non-tuberculous and tuberculous (see page 374). Non-tuberculous osteomyelitis most frequently affects the long bones of the lower extremity (femur and tibia), less often the other long bones, and exceptionally the short bones of the body. In most instances it is the result of infection of the medullary tissue by pus microbes, especially the staphylococcus and streptococcus, which enter the blood from suppurating wounds of the skin (pustular eruption!) or pathologic foci in the respiratory or alimentary tract. As predisposing and contributory causes we may mention the various contagious and infectious diseases, such as typhoid, scarlatina, measles, pneumonia, sepsis neonatorum, etc., all of which being instrumental in lowering the vitality and resistance of the patient.

Violent course.

Diaphyso-epiphyseal separation.

Infection of the medullary tissue once established, the pathologic process is very acute and violent. If left alone the inflammatory process rapidly goes on to suppuration, leading to loosening of the periosteum and bone necrosis and separation of the diaphysis from its epiphysis. If the patient survives and the inflammatory process subsides, there is a separation of the dead bone (sequestrum) from the living. Unless removed the sequestrum may remain an everlasting source of irritation and suppuration.

The osteomyelitic process is ushered in by a chill, rapid rise of temperature and pulse and other symptoms which usually accompany acute suppurative affections. Before the appearance of the local symptoms the disease is very apt to be mistaken for a pyemic or typhoidal condition, and in infants unable to indicate the presence of local pain osteomyelitis may end fatally before a correct diagnosis has been arrived at. Hence the importance of a careful examination of the bony system in all febrile affections with indefinite source..

Symptoms of suppuration.

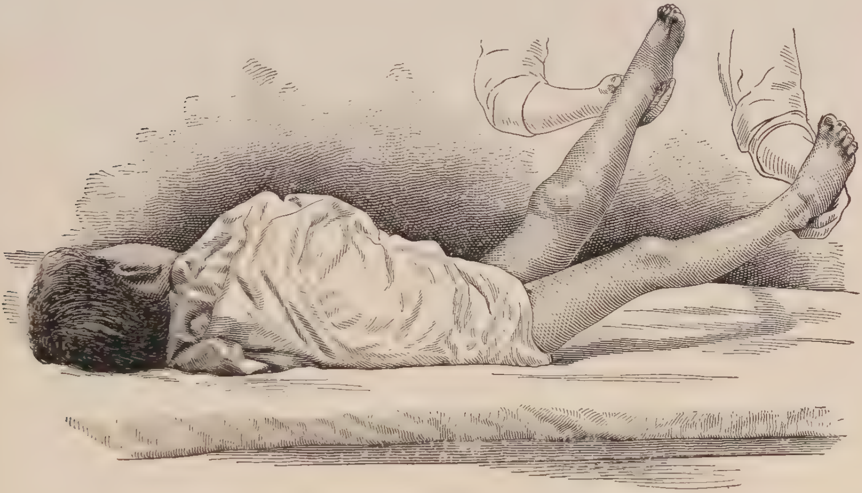

Fig. 122.—Osteomyelitis of Tibia (2 weeks old). Complicated by Extension of Phlegmonous Inflammation to the Prepatellar Bursa. (*Senn.*)

The local symptoms of osteomyelitis are: Pain, tenderness, swelling, redness, synovitis, epiphyseolysis, and loss of function.

The pain is excruciating, boring or throbbing, worse at night, and increases in intensity as the exudation becomes more abundant. Young children are rarely capable of locating the exact seat of the pain, but usually refer to the entire affected limb. As a rule, the pain disappears suddenly with the escape of the inflammatory products from the interior to the exterior of the bone.

Excruciating pain.

Tenderness on pressure can be detected early, and is most severe where the inflammation has approached nearest the surface of the bone. Where the disease is located deeply in the medulla, tenderness can readily be elicited by percussion.

Tenderness on pressure.

Edema.

Swelling and redness are not discernible until the inflammation has reached the periosteum. Thrombophlebitis and edema, however, are often early symptoms.

Pus.

Synovitis is the rule where the disease affects the epiphysis as well as the end of the diaphysis. The intraarticular effusion is at first serous, the result of vascular disturbance, but as the suppurative process in the bone advances, the effusion becomes purulent by direct extension of the infection. The character of the effusion can readily be determined by exploratory puncture.

Epiphyseolysis.

Epiphyseolysis, or separation of an epiphysis from the diaphysis, is a late symptom, or rather a complication. It may be recognized by soft crepitation between the separated parts, false point of mobility and displacement—signs of fracture.

Contracture.

Loss of function of the limb is invariably present, and as the disease advances there are marked contractures. The patient instinctively assumes such postures as will best relax the muscles and ligaments connected with the affected area, and thus prevent painful tension.

Leucocytosis.

These symptoms if closely kept in view will generally avoid errors in the diagnosis. Typhoid fever can readily be excluded even before the development of local symptoms by the presence of marked leucocytosis in osteomyelitis. For differential points between osteomyelitis and arthritides, see page 420.

Embolism.

As previously indicated the course of the disease varies with the degree of infection and the aggressiveness of the treatment. Early operative interference is usually followed by recovery in the great majority of cases. In some cases the infection is extremely violent and death occurs within the first thirty-six hours, before (or in spite of that) a diagnosis had been made and the appropriate therapeutic measures employed. The great danger in osteomyelitis is the tendency to venous and arterial thrombosis with secondary embolism and abscesses in different parts of the body, especially the lungs, heart and kidneys.

With subsidence of the acute symptoms, the osteomyelitic process is not always at an end. Transition into *chronic* osteomyelitis is not uncommon (for details see treatise on surgery). Suppurating sinuses leading down to the infected sequestra may indefinitely persist, and, with occasional improvement, continue to undermine the vitality of the patient. Amyloid disease of various viscera (liver, *q. v.*) may form a sequel of prolonged suppuration.

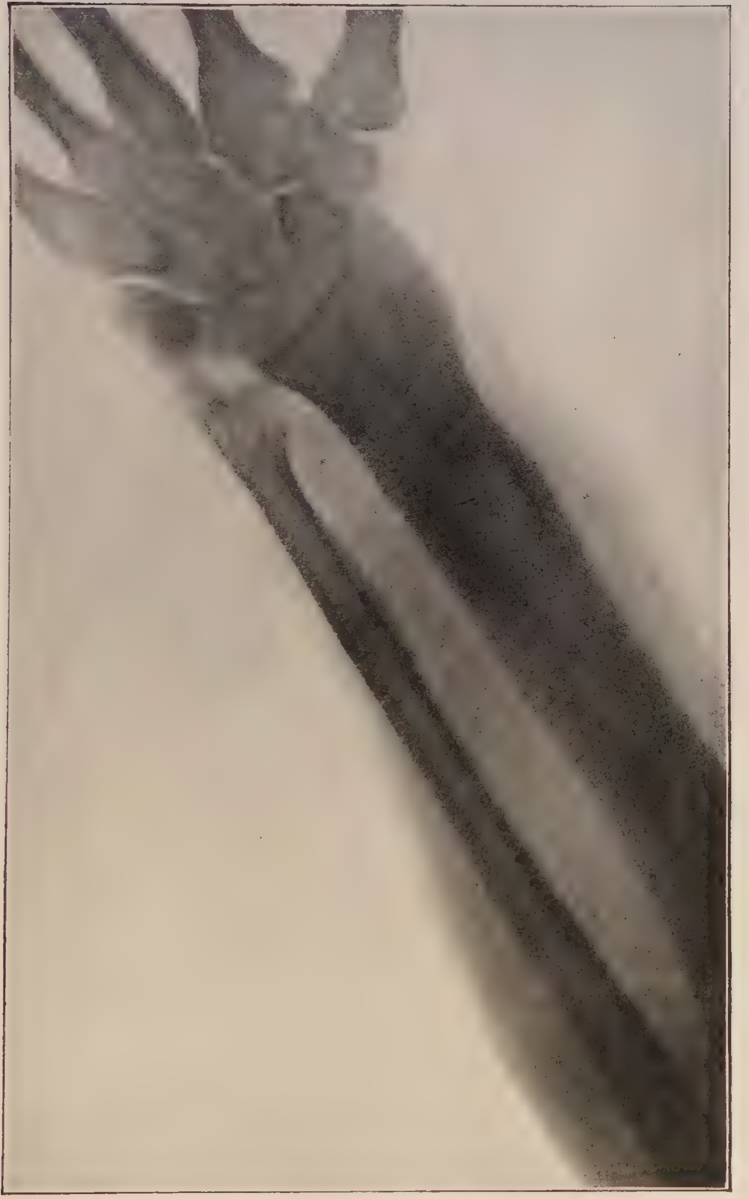

Fig. 123.—Osteomyelitis of the Radius. Enlargement of the entire bone and three well-defined abscess-cavities. (*Senn.*)

SYPHILIS HEREDITARIA S. CONGENITA

(Syphilis Embryonalis or Fœtalis, Syphilis Neonatorum, Syphilis Hereditaria Tarda).

Spirochæte pallida. Congenital syphilis is due to a specific micro-organism, the spirochæte pallida, which is transmitted to the embryo or fetus either through the syphilitic semen (ex patre), ovule (ex matre), or maternal blood (at any time during pregnancy).

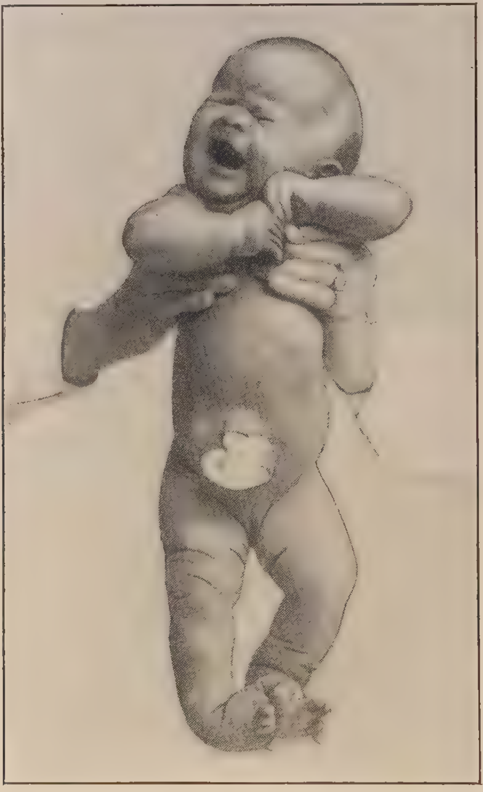

Fig. 124.—Congenital Syphilis (3 weeks old). Note peculiar deformity of feet, excoriation of upper lip, tumefaction on forehead. (*Sheffield.*)

Few survive. The great majority of syphilitic embryos or fetuses are aborted. The few that survive may pass through the syphilitic process in utero (syphilis embryonalis or fœtalis) and emerge into the world either dead or in a shriveled, shrunken, emaciated or disfigured (hydrocephalus, spina bifida, etc.) condition, and, as a rule, succumb soon after birth; or the fetus may maintain a good state of health during intra-uterine life, be born in apparently perfect health, and develop the syphilitic manifestations soon after birth (*syphilis neonatorum*), or not until several years after (*syphilis hereditaria tarda.*)

Having fully discussed "syphilis embryonalis" in connection with "feeble vitality of the newly born" (*q. v.*), we will limit our present remarks to syphilis of the newly born and to late syphilis.

SYPHILIS NEONATORUM.

As previously alluded to, the infant may at birth appear perfectly healthy. It may continue to thrive, especially if fed on breast milk. Before long, however,—usually after from about one week to three months—the clinical aspect changes materially. The baby begins to breathe noisily, especially while it nurses, "sniffles," becomes hoarse, or loses its voice entirely. The nurse or the weather is blamed for the baby's "cold in the head," until examination reveals that the syphilitic coryza is associated with swelling of the nasal mucous membrane and occlusion of the anterior nares by a seromucous or serosanguinolent discharge and incrustation. Inspection of the mouth and throat often discloses grayish-white patches (plaques muqueuses) upon the mucous membrane of the mouth and pharynx, more rarely papillomatous vegetations, and occasionally edema glottidis, which latter may lead to fatal termination. Not rarely the inflammation of the nasal mucous membrane extends to the nasal

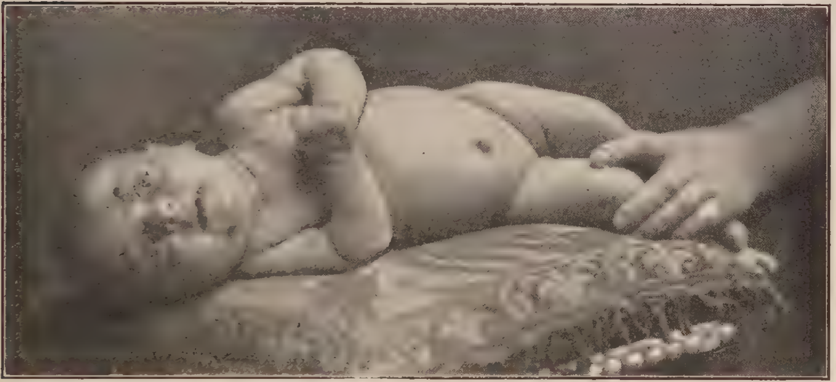
Fig. 125.—Congenital Syphilis (6 weeks old). Note maculo-papular eruption. (*Sheffield.*)

Noisy breathing; ozena.

Mucous patches.

periosteum and perichondrium, arresting the development of the nasal bones, and giving rise to the peculiar sinking of the bridge of the nose which is generally designated "saddle nose."

Saddle-shaped nose.

The syphilitic manifestations augment from day to day. The skin assumes a peculiar light- or dark-yellow (copper) color, is dry and hard to the touch, and soon becomes covered by an eruption which is typical for its multiplicity and variability. Almost every kind of skin disease is represented. Papules, vesicles, pustules, smooth and scaly patches, tubercles, wheals, macules, hemorrhagic spots, simple redness, scabs, ulcers, etc., vie with one another in their supremacy, and rhagades surround the different external orifices of the body (angles of the eyelids and lips, at the alæ nasi, anus, labiæ vaginæ, etc.). The hairy portions of the body also participate in the syphilitic process. The hair of the scalp, eyebrows and eyelashes rapidly fall out and are very slow in returning. The nails undergo certain alterations, such as thickening, claw-like deformities, suppurative inflammation (onychitis) and exfoliation (paronychia), the process not rarely extending also to the phalanges (syphilitic phalangitis, *q. v.*). In the majority of cases we find a bullous eruption which is pathognomonic of grave syphilitic infection, *i.e.*, pemphigus syphiliticus. It usually sets in within the first week after birth as flaccid, yellow or brownish vesicles, surrounded by an areola of dry epidermis or excoriation. The bullæ vary in size from a pinhead to a cherry, burst readily and

Skin lesions.

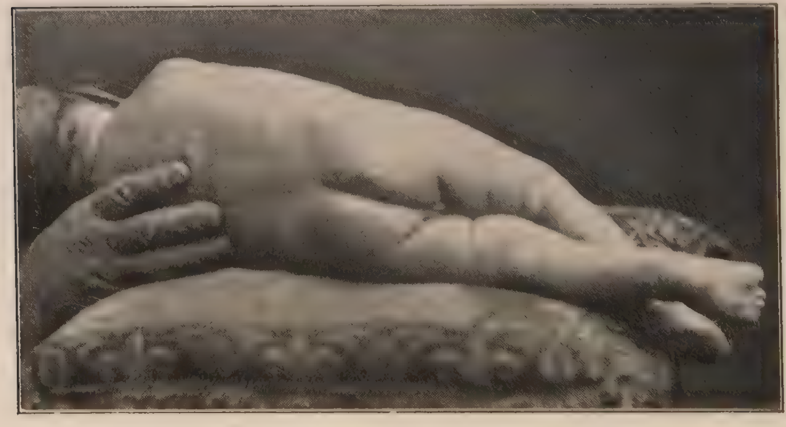

Fig. 126.—Congenital Syphilis. Same case as Fig. 125. Note protruding condylomata at anus. (*Sheffield.*)

Rhagades.

Loss of hair.

Onychitis.

Pemphigus.

discharge a seropurulent or serosanguinolent content. They are distributed all over the body, but particularly over the palms of the hands and soles of the feet—herein differing from non-syphilitic pemphigus which but rarely affects these parts. In consequence of the inflammatory state of the skin the superficial lymphatic glands are more or less implicated, the swelling often persisting long after disappearance of the primary cause. Enlargement of the epitrochlear glands—just above the internal condyle of the humerus is especially common and of diagnostic importance. Special mention deserve also the syphilitic condylomata, especially at the anus and female genitals. They usually begin as simple papules and from the effect of irritating discharges undergo transformation into luxurant growths.

Differentiation from simple pemphigus.

Enlarged epitrochlear glands.

Condylomata.

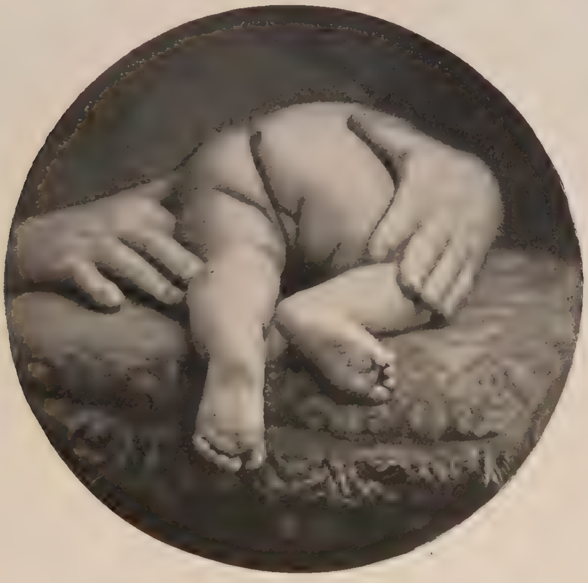

Fig. 127.—Pemphigus Syphiliticus Involving Especially the Soles of the Feet. (*Sheffield.*)

With the aforementioned clinical findings in view, it requires no sage to solve the problem of diagnosis. Now, if the physician bases his judgment upon the symptoms presented, does not allow himself to be led astray by spurious histories (omnis syphiliticus mendax!), but goes right ahead and employs suitable antisyphilitic measures (see page 408), the chances of rapid improvement and ultimate recovery are very good indeed. Otherwise, the syphilitic process often violently runs its destructive course, attacks one structure after another, one organ after the other, crippling the hapless infant for life, if it unfortunately survives.

Osteochondritis.

Pseudoparalysis.

Parrot's nodes.

Deformed forehead.

The osseous system hardly ever escapes involvement. As in fetal syphilis (q. v.), the syphilitic bone affection consists principally of an osteochondritis and sometimes caries and necrosis. There is an overgrowth of the cartilage between the epiphyses and diaphyses of the long bones, often giving rise to painful circular swelling in the epiphyseal region and separation of the affected limb (spontaneous fracture), with consecutive loss of power (Parrot's pseudoparalysis). This process is usually (but not invariably) unilateral—herein differing from rachitis in which the epiphysitis is almost always bilateral. The skull presents enlargements (Parrot's nodes) of the parietal eminences and a buffer-like bossing of the frontal bone which is generally designated as "hot-cross-bun" tumors. Occasionally the frontal bone appears either unduly convex and prominent (front Olympian) or keel-shaped, with a central ridge and lateral flattening. These syphilitic manifestations are often associated with craniotabes, delayed (or premature) closure of the fontanelles and great brittleness of the milk-teeth.

Hepatitis.

The liver is often the seat of cellular infiltration (interstitial hepatitis) or variously sized gummata, rarely large enough to be visible to the naked eye. The liver is enlarged, hard and uneven to touch, but palpable through the abdominal wall only in advanced cases. Marked syphilitic changes in the liver frequently give rise to icterus, acholic stools, and bile-colored urine. On the other hand, mild forms of the disease are usually entirely free from symptoms.

Perisplenitis.

Next to the liver the spleen is most prone to suffer in syphilis. It is enlarged and readily palpable through the abdominal wall. Splenomegaly being of so common occurrence in early childhood, it is difficult to determine how much of this phenomenon is due to the syphilitic process and how much to other causes, especially rachitis. The younger the infant (under six months), the greater the probability of the perisplenitis being syphilitic in nature, especially if the splenomegaly be associated with other syphilitic symptoms, such as "Parrot's nodes," condylomata, and ozena.

Syphilis of the pancreas is not demonstrable during life, but it has repeatedly been proven, by post mortem, that the pancreas is affected in a way very similar to that of the spleen.

The intestines are but rarely affected. Intestinal syphilis is manifested chiefly by ring-shaped indurations of the muscles and mucous membrane, leading to gradual constriction of the intestinal lumen. The pathologic process resembles that of "Peyer's patches."

Intestinal patches.

Clinically intestinal syphilis gives rise to protracted diarrhea, often with fatal termination.

Syphilitic changes (perivascular cellular infiltration; gummatous deposit) are occasionally met also in the kidneys and suprarenals (paroxysmal hemoglobinuria; nephritis), in the heart (symptoms of myocarditis), in the lungs (pneumonia with slow course; spirochæte in the sputum), in the thyroid gland (struma), in the thymus (cyst, or abscess), in the testicles (often greatly enlarged; hydrocele; arrested development), and in the ovaries (demonstrable post mortem; sometimes by rectal, bimanual examination during life).

Changes in urogenital system, lungs, thyroid and thymus.

Arteritis and periarteritis, gummatous deposits and sclerosis occasionally occur in the brain and spinal cord as in the other organs of the body, and the concomitant symptoms vary with the seat of the lesions. Chronic meningitis and hydrocephalus are not rarely of syphilitic origin, and epilepsy, idiocy, local paralysis of the extremities and of the eye muscles, blindness, disseminated sclerosis and tabes dorsalis have occasionally been traced to congenital syphilis. Also cases of syphilitic encephalitis are on record. The resemblance between syphilis of the nerve system and tuberculosis should not be lost sight of.

Chronic meningitis.

Encephalitis.

As already suggested the diagnosis of syphilis is very easy when the aforementioned symptom-complex is in full bloom. Cases, however, are not rarely encountered which are apt to test the skill of even the best diagnostician. I am referring especially to those which either run a very latent course from the beginning, or do so after a few weeks' antisyphilitic treatment. Every bit of information as to the past personal ("snuffles," eruption, etc.) and family history (miscarriages; persistent sore throat in the mother or father!) should be utilized to arrive at a correct conclusion. Old cracks and scars at the anus, mouth, nares, etc.; dark, mottled skin; old marks of healed ulcers in the mouth and throat; persistent ozena; intractable intertrigo, etc.; excessive brittleness of the milk-teeth—should all be carefully looked into, and where doubt still exists the patient be given the benefit of the doubt and actively treated for syphilis—the rapidity

Diagnosis in latent cases.

of response to treatment at the same time serving as a differential point of diagnosis (therapeutic test).

Therapeutic test.

Wherever possible laboratory tests should supplement ordinary clinical examination. Of these Wassermann's serum diagnosis of syphilis is deserving of special consideration (see page 98).

Wassermann reaction.

With establishment of the diagnosis of syphilis, the remedies to be employed to eradicate the disease fortunately leave no room for speculation. The treatment which will be fully outlined in the subsequent pages (see page 408) should be carried out energetically and systematically and continued until apparently every vestige of the disease has been completely removed.

Inadequate treatment not only greatly mars the prognosis of syphilis as to life and recurrences, but only too often is responsible for the development of the symptom-complex which is generally described as "parasyphilis." This group of syphilitic manifestations (syphilitic cachexia) consists of extreme debility, marasmus (especially in artificially fed), profound anemia (pseudoleukemia), obstinate gastrointestinal and bronchial catarrh, otitis (deafness), disposition to rachitis, cretinism and idiocy, and lowered power of resistance to divers acute infectious diseases. While the mortality of the carefully treated syphilitics is comparatively small, those who are carelessly managed often succumb to intercurrent diseases, even of the most trifling character, not rarely die suddenly without apparent cause, and if they survive, remain decrepit for life, and a source of horrible misery to future generations.

Parasyphilis.

High mortality.

SYPHILIS HEREDITARIA TARDA S. LATA.

Late hereditary syphilis attacks the offspring of syphilitic parents at any period between early childhood and adolescence. The children thus affected may or may not have shown manifestations of congenital syphilis during intra-uterine life or soon after birth. The symptoms, however, are more pronounced in those who had been treated inadequately or not at all. Late hereditary syphilis essentially corresponds to the tertiary stage of acquired syphilis. Like the latter it shows a predilection for the osseous system; but no structure or organ of the body is exempt from its destructive effects.

Corresponds to acquired tertiary stage.

As we will presently demonstrate, the lesions of late hereditary syphilis may be numerous and grave, but not always strictly pathognomonic of this disease. There is, however, one group of syphilitic manifestations, which, if present, invariably betrays the presence of a syphilitic taint.

Triad of syphilis.

This symptom-complex is generally described as the "triad of syphilis" and consists of the following phenomena:—

Hutchinson teeth.

1. The so-called Hutchinson teeth. The characteristic teeth of syphilis are the two upper central incisors of the permanent set. The teeth are chalky, ill-developed, small, and irregularly placed. They taper from the free border to the base—hence the term "screwdriver teeth"—and present a broad, semilunar notch in the center of the edge. They should not be confounded with the brittle and decayed milk-teeth observed in infantile syphilis or rickets, and the irregularly implanted teeth associated with deformed palate or dental arches.

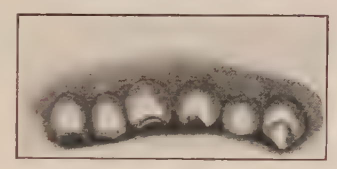

Fig. 128.—Syphilitic, "Hutchinson Teeth." Note semilunar notches in central incisors. (*Sheffield.*)

Keratitis.

2. Interstitial keratitis. The almost invariably symmetrical affection begins with corneal haziness which rapidly increases until the entire cornea is in a condition of partial opacity resembling "ground-glass." It is associated with congestion of the ciliary region and slight inflammation of the conjunctiva, and in severe forms of the disease, with iritis, retinitis and choroiditis. In addition to the corneal gray-colored patches, abruptly margined, crescentic patches of salmon tint are often present on the corneal surface, this sign of vascularity not rarely spreading over the whole cornea and giving rise to a deep plum tint of purple redness. Excessive lacrimation and photophobia prevail from the start, in marked cases reducing the patient to a state of practical blindness. The disease runs a very slow course, from about three months to a year or longer, and when it subsides leaves behind more or less marked corneal opacity and visual impairment.

Deafness.

3. Deafness. This condition is not accompanied by any inflammatory symptoms. It is caused by syphilitic involvement of the labyrinth (often of both ears). The deafness very rarely

clears up spontaneously and entirely. On the contrary, even under active treatment permanent defective hearing is the rule. This peculiar form of deafness often precedes or follows the attack of keratitis and is gradual in its development.

Permanent deafness.

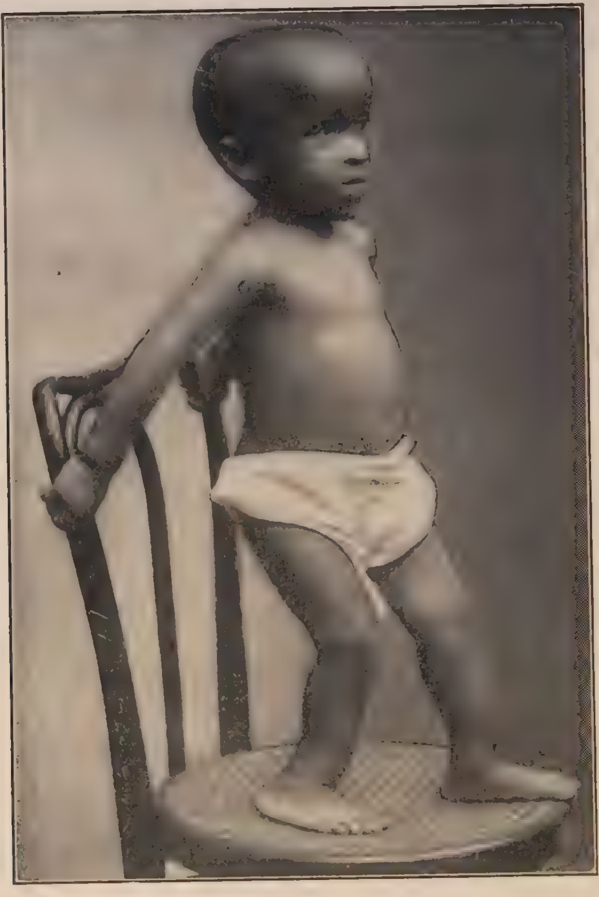

Fig. 129.—Syphilitic Osteoperiostitis of the Tibiæ. "Saber-shaped Deformity." Note also deformed bridge of nose. (*Sheffield.*)

Osteo-periostitis.

The bone lesions of syphilis consist of an osteoperiostitis, or soft gummatous periostitis, especially of the tubular and cranial bones. The most frequent seat of the disease is the tibia; then follow the ulna and radius, the humerus, femur, clavicle, the bones of the skull, the phalanges and sternum. Syphilis of the shaft of the tibia usually gives rise to a characteristic "saber-

Saber-shaped deformity.

shaped" deformity of the tibia, the so-called "tibia en lame de sabre." It differs from the rachitic deformity of the tibia by its crest being rounded (in rickets it is sharpened) and its internal and external surfaces convex (in rickets they are flat or concave).

The cranial bones are affected in a manner similar to that of syphilis neonatorum (see page 402). Ulceration of the soft palate and perforation of the hard palate and nasal bones with secondary "saddle-shaped" deformity of the nose are of common occurrence.

Saddle-shaped nose.

Dactylitis.

Syphilis of the phalanges (syphilitic dactylitis) is characterized by a puffy, fusiform, or spindle-shaped swelling. It affects the fingers more often than the toes. The inflammation may begin either in the connective tissue and ligaments or in the periosteum and bone. If left alone the disease progresses rapidly and leads to protracted osteomyelitis with ankylosis, shortening and permanent deformity of the affected parts. Syphilitic dactylitis differs from the tuberculous variety, which it greatly resembles, by its being less common, often symmetrical and accompanied by other syphilitic lesions.

Differentiation from tuberculosis.

Synovitis.

Occasionally the joints participate in the syphilitic process, but the affection is rarely of serious nature. It essentially consists of a recurrent synovitis with thickening and ankylosis, and may readily be mistaken for articular rheumatism. The absence of fever and redness and the history of syphilis usually clear up the diagnosis (see page 419).

Subcutaneous gummata.

The skin sometimes presents subcutaneous gummata which when neglected have a great tendency to break down and to form large phagedenic ulcers. They are most frequently met on the face and upper part of the thighs or legs. They promptly yield to energetic antisyphilitic treatment—a feature to be borne in mind in the differential diagnosis between syphilitic and tuberculous ulcers.

Similar ulcerating gummata are not rarely found in the mouth, nose and throat. If not promptly arrested they are rapidly destructive and may occasion extensive disfigurement.

The lymphatic system and the viscera, especially the liver and spleen, rarely fail to show late syphilitic manifestations. The latter are essentially identical with those described in connection with congenital syphilis neonatorum (see page 402).

Finally, mention may be made of the tendency of late syphilis to arrest the development of the child's body and mind. Dwarfism and infantilism are not rarely traceable to this baleful

Infantilism.

cause. Indeed, appreciating the gravity, multiplicity and complicacy of the syphilitic lesions, it is rather surprising that the aforementioned bodily and mental deteriorations are not more rampant.

Notwithstanding the apparent explicitness of the symptomatology, the diagnosis of late hereditary syphilis is by no means a simple proposition. It is especially difficult in cases complicated by intercurrent diseases, *e.g.*, tuberculosis or rickets.

Pathognomonic symptoms.

The specific history; the simultaneous occurrence of lesions in various parts of the body; the tendency of the bone lesions to be symmetrical; the appearance of the manifestations very frequently in the midst of apparently perfect health, and, finally, the quick response to antisyphilitic treatment—are more or less decisive in the diagnosis. Of course, all doubt is removed by positive microscopic or bacteriologic findings, especially serum diagnosis.

ACQUIRED SYPHILIS.

Contagiousness of syphilis neonatorum.

Newly born infants may acquire syphilis either intrapartum, by coming in contact with a chancre in the parturient canal, or while nursing of the breast of a woman (mother or wet-nurse) in the contagious state of syphilis. The disease may further be acquired by infants and older children practically in the same manner as by adults. It is well to remember that newly born infants with secondary symptoms of syphilis may transmit the disease to healthy people through fondling, the use of articles coming in contact with syphilitic lesions, etc. I have now in mind two older, previously healthy brothers who have in this manner acquired syphilis from a newly born syphilitic child.

The course of acquired syphilis in children is identical with that observed in adults, except that it is prone to be more rapid and violent.

Mercury.

Treatment of Syphilis.—The treatment of syphilis is alike in both forms of the disease—inherited (early and late) and acquired. It should be begun with as soon as the diagnosis has been established. Temporizing is often fatal. Mercury in some form is the only remedy that is certain in its results, and should be administered *continuously* until every vestige of the disease has apparently disappeared, and then given *at intervals* of from two to six weeks for a total period of from two to three years. Calomel is the preparation *par excellence.* One-tenth to one-fourth of a grain twice (to an infant) or thrice (to an older child) will usually suffice. Now and then we may also employ sodium iodid (half a grain for every year of the child's age) three times a day, or the syrup of the iodid of iron (three drops for an infant under one year, five drops for two years, and ten drops for over five). To hasten saturation of the system with the mercury, we may, *in addition,* resort to mercury inunctions. From 10 to 30 grains of mercurial ointment may be rubbed in once a day alternately into the axilla, groin, abdominal wall, calf-muscles, and loins. To prevent excessive salivation the oral cavity should twice daily be washed with a 2 to 5 per cent. solution of chlorate of potash or tincture of myrrh. Syphilitic ulcers should be cauterized with nitrate of silver solution (3 per cent. to 10 per cent.). Keratitis calls for local use of atropin sufficient to keep the pupils widely dilated, hot poultices (by means of moist hot cloths), occasional dusting of calomel over the corneal ulcers, protection from bright light (dark room or smoked eye-glasses), and, of course, internal administration of mercury and the iodids. The great majority of cases of osteitis yield promptly to constitutional treatment, but where necrosis is pronounced the management must follow ordinary surgical lines. Persistent condylomata will rapidly disappear after a few applications of a 5 per cent. salicylic-resorcin-collodion solution, or occasional painting with caustics. Onychia and paronychia should be treated by local bichlorid baths (1 : 2000), once or twice daily, and dusting with calomel 1 part, gum arabic 1 part, and stearate of zinc 20 parts. Indurated lymph glands usually yield to potassium iodid ointment, while suppurating glands require surgical interference.

The iodids.

Salivation.

Ulcers.

Keratitis.

Osteitis.

Condylomata.

Onychia.

Adenitis.

The general health of the patient should not be lost sight of. Other conditions being favorable, a syphilitic mother should nurse her syphilitic child. This being impossible, the infant should be put on properly modified cows' milk, or on the breast of a wet-nurse who emerged from an attack of syphilis without serious consequences. In older children also particular attention should be paid to good nutrition. The tendency of rickets complicating syphilis should be borne in mind. Hydrotherapy, plenty of fresh, pure air, and general tonics are essential to success.

Maternal nursing.

Within the last few months marvelous cures have been reported from the hypodermatic use of dioxydiaminoarsenobenzol (the mysterious "606" of Ehrlich and Hata). The remedy is dissolved in commercial soda lye by rubbing in a mortar; glacial acetic acid, drop by drop, is then added, obtaining a fine yellow precipitate. This is suspended in distilled water and the reaction

606.

of the liquid is made exactly neutral to litmus paper. The suspension (containing from 2 to 5 grains of the remedy) is slowly injected below the shoulder blade or in the gluteal region. One injection is claimed to arrest the disease.[1]

MALARIA

(Febris Intermittens. Febris Remittens, Æstivo-autumnal).

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od, free or within the red
puscles of parasites
asmodia) that exert a
eterious influence
on the red cells.

Malaria is endemic in the greater portion of the inhabited world, and is most prevalent in moist tropical regions. No age is exempt from this disease. The exciting cause of malaria is the hematozoön of Laveran conveyed to the human body principally by the bite of the Anopheles mosquito which has previously sucked the blood of a malarial patient and has acted as an intermediate host for the malarial parasite. The hematozoön enters the blood-corpuscles and, after undergoing the different stages of development, the blood-current—at this time giving rise to the characteristic chill or paroxysm. Depending upon the period of maturity and species of the plasmodium, the febrile attack may occur every day (quotidian); every two days, going on the third (tertian); every three days, going on the fourth (quartan) day; or may be more or less continuous with daily remissions (remittent or estivo-autumnal fever).

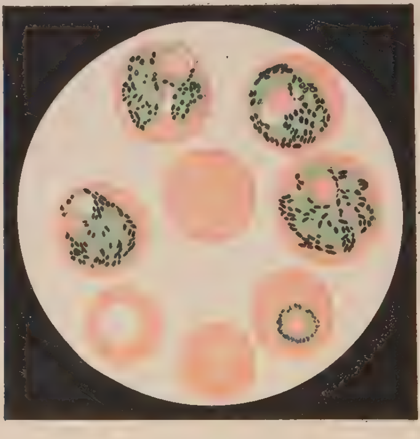

Fig. 130.—Malaria Plasmodia; Tertian Type. Plehn-Chenzinsky's Stain. × 1000. (*Lenhartz* and *Brooks.*)

Transmission through mosquito bites.

Quotidian, tertian and quartan types.

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[1] Syphilitic newly born infants who are nursed by their mothers (or syphilitic wet-nurses) will derive the full benefit of the remedy by administering it to the mother. In fact, this method of treatment is greatly to be preferred to direct treatment of the baby.

INTERMITTENT FEVER.

This form of malaria is characterized by the occurrence, at regular intervals, of paroxysms divided into four stages—premonitory, chill, fever, and the sweat. During the premonitory stage the patient complains of headache, lassitude, and nausea; he vomits, yawns, is irritable and drowsy. Suddenly he is seized with a feeling of cold—the chill. Chill. The features become pinched, the lips blue, the skin cool and rough (*cutis anserina*); he shivers and shakes, and his teeth chatter while the thermometer in the axilla or rectum shows a decided rise of temperature. These phenomena may continue for from a few minutes to an hour or longer and are then gradually replaced by those of the hot stage, *i.e.,* hyperpyrexia, flushed face, headache, full pulse, intense thirst, scanty urine, sometimes nausea, vomiting and severe nervous manifestations. Fever. The hot stage lasts from three to six hours or longer, and subsides gradually, being succeeded by more or less marked sweating, rapid defervescence and abatement of the other symptoms. Sweat. The duration of the entire paroxysm is from six to twelve hours, after which time the patient is apparently well—until the return of a new attack, which, as already mentioned, may occur every day, every two days or three days.

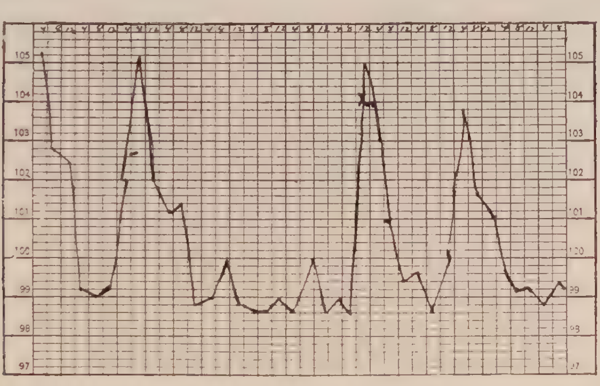

Fig. 131.—Temperature Chart of Quotidian and Tertian Malarial Fever in a Child 22 Months Old. The fever changed from quotidian to tertian on administration of a few large doses of quinine. (*Sheffield.*)

This description corresponds with the symptomatology of typical intermittent fever, uninfluenced by medication, as it occurs in children over ten years of age. It is thus identical with that in adults. In younger children the course of the paroxysms pre-

Deviations in infants.

Cyanosis.

Enlarged spleen.

sents numerous deviations. The prodromic and cold stages may be absent or of very brief duration. The chill may be replaced by grave nervous manifestations, such as convulsions, or be indicated only by cyanosis of the lips and the tips of the fingers and toes. Sweating is slight or absent, or may be well marked and continue until the subsequent paroxysm of fever. Young children are rarely entirely free from discomfort during the intermittent stage. As a rule, they are exhausted, restless, have no appetite, etc. With repeated attacks of the fever there is marked swelling of the spleen and great diminution in the number of red blood-cells.

Differentiation from tuberculosis, pyemia, typhoid and influenza.

In view of the aforementioned deviations from the typical course of the paroxysms, the diagnosis of intermittent fever in young children often presents great difficulties. It is apt to be mistaken for tuberculous (meningitis, lymphangitis, peritonitis, etc.) and pyemic (empyema, pyelitis, ulcerative endocarditis, otitides, etc.) processes, typhoid and influenza. A correct diagnosis, however, can usually be arrived at by exclusion, always bearing in mind the facts that in malaria the plasmodium malariæ or secondary pigmentation of the blood-cells is invariably present in the blood and that the course of the disease is greatly modified by full doses of quinine. The finding of an enlarged spleen (also liver) without a history of syphilis or rickets points to malaria of more or less prolonged standing, and a history of malaria is, of course, corroborative in the diagnosis.

REMITTENT (ÆSTIVO-AUTUMNAL) FEVER.

Occasionally epidemic.

Continued fever, with irregular remissions.

This type of malarial fever is usually observed in temperate zones, principally in the autumn. In institutions where large numbers of children are congregated it may occur in epidemic form and lead to grave diagnostic errors. It usually sets in suddenly with malaise and chilliness, followed by fever with exacerbations and remissions, the temperature during the latter, however, remaining constantly above normal. The other symptoms are very indefinite. As in all febrile diseases, anorexia, nausea, sometimes vomiting, headache, drowsiness and lassitude predominate. In some cases gastrointestinal symptoms prevail, in others respiratory. But the cardinal manifestations of the affection are the continued fever of from one to three weeks' duration with irregular remissions, palpable spleen, and the plasmodium malariæ in the blood. Bearing these clinical symptoms in mind and those of the diseases suspected, there ought to be no difficulty in differentiating remittent fever from typhoid fever or protracted influenza—with both of which diseases it is most apt to be confounded. The quinine test is not reliable in the remittent form of malaria.

The prognosis of remittent fever is favorable, except for the tendency to recurrence at shorter or longer intervals and of ultimately becoming chronic.

CHRONIC MALARIAL CACHEXIA.

The diagnosis of this condition is often very difficult, since its principal symptoms—anemia, debility, enlarged spleen and liver—are also pathognomonic of severe rachitis, pseudoleukemia, and similar wasting diseases. Corroborative data may be obtained from a history of previous attacks of either intermittent or remittent fever or the occurrence of periodical headache, neuralgia, dysentery or hematuria. One should be very cautious, however, in making a hasty diagnosis of "malaria," unless there be ample reason for exclusion of the other affections and the therapeutic quinine test prove positive.

Periodic headache, hematuria, etc.

Chronic malarial cachexia *per se* is not dangerous to life, but is apt to prove so from its concomitant symptoms, such as profound anemia and amyloid degeneration of the viscera.

Treatment.—As malarial fever is ordinarily contracted through the bites of mosquitoes, to prevent malarial disease, we must either destroy the mosquitoes or avoid their bites. An effort should be made also to isolate, by mosquito-netting, all cases of acute malarial disease, in order to deprive the mosquitoes of the infective material. Another very important measure is to prevent the breeding of the mosquitoes. Mosquitoes lay their eggs in water-barrels, pans, tin cans, pots, kettles, wells, springs, rainpools, cesspools, drainage traps, ponds—in short wherever stagnant water is found. We have to see to it that all water receptacles are closely covered with thin, wire gauze, and that where drainage cannot be carried out, the surface of ponds, etc., is covered with a film of kerosene oil. One ounce of oil to fifteen square feet of water will usually suffice. The oil must be renewed about once a week during the mosquito season. A solution containing one pound of sulphate of copper and one pound of unslaked lime in ten gallons of water will kill the mosquito larvæ when added in the proportion of one of the solution to fifty of the infected water.

Destruction of mosquitoes.

Kerosene oil.

White people settling in malarial tropical regions should not plant their houses near native settlements.

Where the aforementioned prophylactic measures cannot properly be enforced, resort should be had to the routine administration of quinine during the mosquito season.

Quinine specific.

By mouth.

Per rectum.

Hypodermatically.

Whether as a prophylactic or curative measure, quinine is the specific destructive agent of the malarial parasites. To obtain prompt results it should be given in full doses. Children tolerate relatively much larger quantities of quinine than adults. An infant of two years requires about 15 or 20 grains a day until the attack is controlled and smaller doses after. For children unable to take quinine in capsules I prefer the newer "tasteless" quinine preparations, such as quinine ethyl carbonate, diquinine carbonic ester, etc., or administer (see page 116) the ordinary bitter quinine per rectum (quinine subsulph. gr. v in ℥ij of water by means of colon tube). In cases of marked gastric irritability or in those very grave in nature or protracted in course, quinine may be employed in 5 gr. doses hypodermically. For this purpose bimuriate of quinine and urea, the hydrochlorosulphate, the hydrobromate, or the bisulphate may be used. Ugly sloughing which is apt to follow at the site of the injection may be prevented by cleanliness of the needle and skin and by throwing the solution deeply into the subcutaneous tissues and sealing the point of puncture with adhesive plaster.

Iron and arsenic.

In protracted cases iron and arsenic (Fowler's solution) will be found useful additions to the quinine, and when there is a great tendency to recurrences, permanent residence in dry mountainous regions will sometimes remain the only curative measure at our command.

℞ Quinine ethyl carbonate, or diquinine carbonic ester...	ʒss	2
Syr. simplicis	q. s. ad ℥ij	60

M. Sig.: ʒj every two to four hours for a child 3 years old.

℞ Quininæ mur.	gr. xv	1
Acetanilidi	gr. vj	0.4
Podophyllini	gr. ⅛	0.008
Ext. nucis vomicæ	gr. ¼	0.016

M. ft. caps. no xij.
Sig.: Two capsules every three hours for a child 10 years old.

℞ Acidi arseniosi	gr. 1/10	0.006
Quininæ mur.	ʒss	2
Ferri sulph. ex.	gr. x	0.666
Pulv. rhei	gr. v	0.333

M. ft. caps. no. xx.
Sig.: Two capsules every six hours for a child 10 years old. (Chronic malaria.)

℞ Elixir ferri pyrophosphatis, quininæ et strychninæ
(N. F.) .... ℥iss | 45
Syr. aurantii ....q. s. ad ℥iij | 90
M. Sig.: ʒj three times a day for a child 4 years old. (In convalescence.)

RHEUMATISMUS ACUTUS

(Rheumatic Fever, Polyarthritis Acuta).

Acute inflammatory rheumatism is an infectious disease with a specific predilection for the fibrous tissues and serous membranes. The muscular and neural structures, however, are not exempt from it. The discovery of the rheumatism-producing micro-organism is a matter probably of the very near future.

Microbic origin.

Like other infectious diseases rheumatic fever is most prevalent in certain climates and seasons of the year. It presents a prodromic stage of variable duration, which is characterized by chilliness, languor, etc. Like the eruptive fevers it is manifested by general febrile disturbances with local lesions. To a certain extent it is self-limited, since with exhaustion of the fertile soil in one place, the inflammation "jumps" to another place. It ordinarily yields promptly to specific medication—in this respect, also resembling infectious fevers, *e.g.*, malarial fever.

After a brief prodromic stage, the symptoms of acute rheumatism usually set in suddenly, with chills, rise of temperature, vomiting, and vague pain in several parts of the body. In very young children the onset is not rarely associated with cerebral symptoms, especially convulsions. Older children often complain of sore throat, and in some cases articular swelling forms the first and principal manifestation of the affection. The disease once established differs in its symptomatology and course but little from that observed in rheumatism in adults, except, as will be seen later, that in children there is a great tendency toward cardiac complications, while the articular involvement is usually less pronounced.

Sore throat.

Stiffness, redness, swelling and pain.

The joints of the knee, ankle, elbow and wrist are most commonly affected, occasionally also those of the phalanges and hip. The articular involvement is accompanied by stiffness, slight redness, swelling and excruciating pain, the latter especially on moving or handling the parts affected. The inflammation may abruptly cease at one or more joints and, as suddenly, attack others. During the acute stage the temperature ranges between 102° and 104° F., and as the inflammation "jumps" from joint

Jumping from joint to joint. to joint there is usually a sharp rise of temperature. Correspondingly the temperature falls with abatement of the local manifestations. The urine is usually scanty and high-colored, filled with urates, and occasionally contains traces of albumin. **Rarely sour sweat.** The characteristic sour (lactic acid) sweats observed in adults are much less pronounced in children.

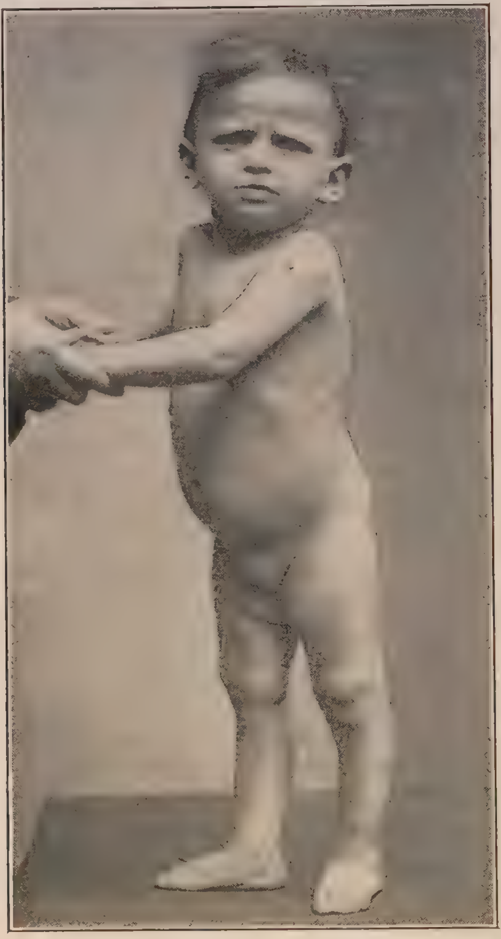

Fig. 132.—Rheumatic Polyarthritis (2 years old). Note swelling of knees; tumefactions at right ankle and foot, effacing the normal bony prominences and the arch. (*Sheffield.*)

There is no definite limitation to the duration and course of the affection. Mild cases, after pursuing a few days, may either recover entirely or enter into a subacute, afebrile stage, and for weeks and months be manifested by vague articular and muscular pain, and ultimately end either in complete recovery, or leave behind some form of subacute or chronic heart disease. Indeed, it is usually in such cases that the heart affection is overlooked, and accidentally discovered some time (years!) later, without being able to disclose a rheumatic history. Severe cases may run a febrile course of from three to five weeks, and if left untreated, sometimes, as many months. It is well to remember that the gravity of an attack is not always commensurate with the severity of the articular involvement. In quite a number of cases endocarditis or pericarditis, or both, may predominate while the other symptoms are barely noticeable. Hence the importance of a routine and careful examination of the heart of children suffering from rheumatic and "growing" pain, or chorea. The latter disease, by the way, is closely allied to, and may precede, accompany or follow rheumatism in its various forms. (See "Chorea.")

Heart disease often overlooked.

Rheumatism and chorea.

Endocarditis.

The earliest symptoms of rheumatic endocarditis are increase of frequency and intensity of the heart-beat and precordial pain. This is soon followed by the usual physical signs of endocarditis —those of mitral regurgitation predominating. Endocarditis forms the most frequent (in about 60 per cent.) complication of inflammatory rheumatism and usually sets in within the first ten days from the onset.

Pericarditis.

Pericarditis is observed only in about 10 per cent. of the cases, and somewhat later than endocarditis. It is manifested by a serous exudation which may rapidly, and unnoticeably, disappear, or persist and lead to pericardial adhesions and their accompanying more or less grave sequelæ.

Pleuritis and pneumonia.

Peritonitis and nephritis.

Less frequent complications are pleuritis and pneumonitis. Both of these affections are ordinarily limited to the left side. The pleuritic effusion may be serous or serofibrinous and is most frequently associated with pericarditis. Of still less frequent occurrence are peritonitis and nephritis. The abdominal pain, however, not infrequently complained of by children during an attack of rheumatism is usually due to muscular hyperesthesia and not to peritoneal involvement.

Muscular rheumatism.

Torticollis.

As in adults, rheumatism of children may also affect the muscles. Rheumatic torticollis is especially common, and in severe cases is apt to be mistaken for cervical spondylitis. Muscular rheumatism affecting the muscles of the lumbar region may resemble lumbar spondylitis; and that of the leg may give rise to symptoms (pain on motion, lameness, stiffness, etc.) simulating coxitis. As previously mentioned rheumatism of the abdominal muscles may simulate peritonitis, while rheumatism of the intercostal muscles may be mistaken for dry pleurisy. In all these cases a diagnosis can usually be arrived at by bearing in mind the pathognomonic symptoms of the affections the muscular rheumatism resembles, and the facts that the latter promptly yields to the salicylates, and that there, as a rule, is a history of involvement of other groups of muscles.

Resemblance to spondylitis and coxitis.

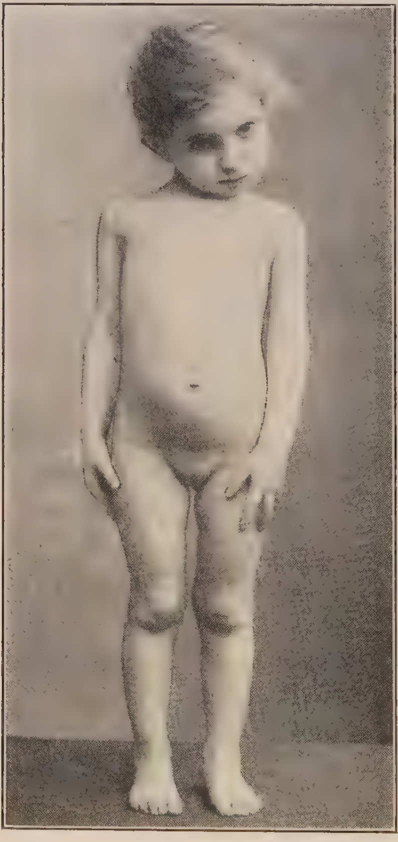

Fig. 133. — Rheumatic Torticollis in a Child 6 Years Old, which greatly resembled Cervical Spondylitis. (*Sheffield.*)

Differentiation from osteomyelitis.

Rheumatism may also affect the periosteum and give rise to thickening of the underlying bone, which condition, with the accompanying pain and fever, may simulate incipient osteomyelitis. From what has been said, it can readily be seen that the diagnosis of rheumatism in its various phases is far from being easy.

Moreover, articular rheumatism may also be mistaken for: Syphilitic, gonorrheal, tuberculous, and the so-called septic arthritides, scurvy, and its allied affections and osteomyelitis.

Pathognomonic symptoms.

In our endeavor to differentiate rheumatism from the divers forms of articular and periarticular inflammations we must bear in mind that rheumatism is a primary febrile affection, as a rule, sudden in development; that its inflammatory process is transient, and its localization multifarious and rapidly shifting, and, finally, that its course is promptly and often permanently influenced by the salicylates.

Differential diagnosis.

Epiphysitis Syphilitica.—Develops slowly, in the first few months of life—rather exceptional for rheumatism—in association with other symptoms of congenital syphilis. It runs an afebrile course and yields promptly to antisyphilitic medication.

Slow onset, afebrile course.

Arthritis Heredosyphilitica (Tarda).—Develops gradually, and affects principally one or both knees. It is usually associated with other syphilitic symptoms, especially parenchymatous keratitis. As a rule, the subjective disturbances are incongruous with the severity and extent of the local signs, and the arthritis is but rarely accompanied by inflammatory symptoms. It yields promptly to antisyphilitic medication. Puncture of the swelling reveals serofibrinous fluid and not rarely the spirochæte.

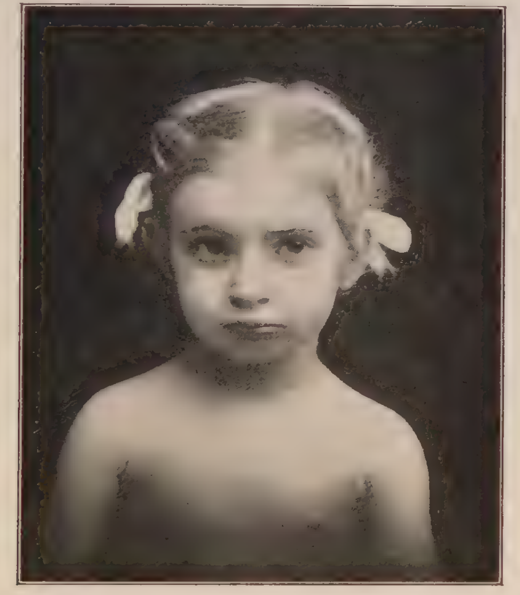

Fig. 134.—Same case as Fig. 133. Three weeks later. (*Sheffield.*)

Mild subjective symptoms.

Spirochæte.

Arthritis Gonorrhœica.—It occurs as a complication of gonorrheal ophthalmia, urethritis or vulvovaginitis. It is most frequently limited to one knee, more rarely to both knees, or the maxillary or sternal articulations, and is accompanied by pronounced inflammatory local and general symptoms. The articular

Gonorrheal history.

involvement is more lasting than that of acute rheumatism, and resists antirheumatic measures.

Arthritis Tuberculosa.—It develops gradually, usually remains limited to one joint, and resists antirheumatic treatment. Atrophy of the affected limb sets in early, and an X-ray examination often shows involvement of the bone. Tuberculin reaction often positive.

Atrophy.

Tuberculin test positive.

Arthritis Septica.—Septic or infective arthritis arises secondarily to sepsis (*e.g.*, purulent arthritis, in sepsis neonatorum) or acute infectious diseases, such as typhoid fever, influenza, pneumonia, diphtheria, scarlatina, etc. The history is the most reliable clue in the diagnosis, and the finding of the streptococcus, pneumococcus, etc. in the seropurulent fluid obtained by exploratory puncture of the swelling is decisive.

Secondary.

Scorbutus (Barlow's Disease), Purpura Hæmorrhagica and Hemophilia (with sanguineous effusion into the joints) also may be mistaken for acute articular rheumatism. In the hemorrhagic diseases, however, there are hemorrhages from and into other parts of the body. The articular swelling is not as evanescent. Fever is usually absent or slight. Furthermore, Barlow's disease is observed in very young infants, who are rarely attacked by rheumatism. Antirheumatic treatment is futile.

Hemorrhages.

Osteomyelitis.—The swelling does not appear until a few days after the onset of the disease, and has its center, not opposite the joint, as in articular rheumatism, but above or below, opposite one or other of the epiphyses of the bones entering into the formation of the joint. In advanced cases the swelling extends along the shaft to a variable distance. In contrast to osteomyelitis rheumatism is rarely limited to a single joint, and its swelling never suppurates. Leucocytosis is absent in rheumatism, and, as a rule, marked in osteomyelitis. A skiagraph is helpful in the differential diagnosis.

Different localization of swelling.

Marked leucocytosis.

Rheumatic fever *per se* is very rarely fatal, but only very few patients emerge uninjured from a severe attack of rheumatism. In probably two-thirds of the cases some form of heart-disease is acquired which sooner or later manifests evidence of its destructive character. This obtains particularly in recurrent rheumatism as well as in cases improperly cared for as regards rest and specific medication.

Prognosis.

Rest in bed.

Rest in bed is the most important therapeutic measure in the prevention of grave complications and sequelæ, and should be

Salicylates. **Iodids.** **Compresses.**

enjoined at least during the febrile course of the disease. Medicinally the salicylates act specifically in all acute rheumatic conditions and their administration should be continued until every vestige of the disease has disappeared. In the beginning the salicylates should be pushed to their full tolerance—say one grain of the sodium salicylate for every year of the child's age, every two hours, until the acute symptoms have been arrested, then every four or six hours according to indications. With the appearance of cardiac complications the iodids, in small doses, should be added, and if necessary also digitalis. For the relief of articular pain and swelling the joint should be enveloped in absorbent cotton wrung out of a warm saturated solution of bicarbonate of soda. The compress should be covered with oiled silk and flannel bandage and changed every two to four hours. When the pain is very acute I found the following very serviceable:—

℞ Olei gaultheriæ, Guaiacolis, Ichthyolis	āā ʒss	2
Adipis lanæ	℥j	30

Sig.: Apply gently twice a day, and cover with flannel bandage.

Fever diet. **Cod-liver oil.**

Acute rheumatism being an infectious disease, I have no faith in "mathematical dietetics" as a cure of the disease, hence do not employ any specific dietary, but limit the diet to a so-called "fever diet" during the febrile stage of the disease and to easily digestible food of all sorts later. This has the advantage of maintaining the nutrition of the patient who at best is weak and anemic. The prolonged use of the iodid of iron and cod-liver oil is always in order in the convalescent stage, and a sojourn in a dry and high inland resort will prevent recurrence and chronicity.

℞ Natrii salicyl.	ʒij	8
Mist. rhei et sodæ	ʒiij	12
Aq. destil.	q. s. ad ℥iij	90

M. Sig.: ʒj every two to four hours for a child 4 years old.

℞ Antipyrinæ	ʒss	2
Natrii salicyl.	ʒiss	6
Caffeinæ natrii benzoatis	gr. xvj	1
Syr. simplicis	ʒiv	16
Aq. destil.	q. s. ad f℥ij	60

M. Sig.: ʒj every six to twelve hours for a child 4 years old, for quick relief of pain.

℞ Olei gaultheriæ	ʒj	4

Ft. caps. no. xij.
Sig.: One capsule every four to six hours for a child 6 years old. (For subacute rheumatism.)

RHEUMATISMUS CHRONICUS.

Chronic rheumatism in children is very rare. As in adults it may supervene after recurrent attacks of acute or subacute rheumatism, or, very exceptionally, it may develop primarily. In either case the local manifestations are clinically alike, and consist of gradual enlargement of the affected joints, painful and hindered motility, ankylosis, and deformity of the bones at the articulations. The course of this form of rheumatism, though very protracted, and extending over a period of years, is usually not as slow as in adults. It eventually leads to crippling of the patients, and fatal termination either from exhaustion or secondary tuberculosis.

Differentiation from syphilitic and tuberculous arthritis.

Chronic articular rheumatism may be confounded principally with syphilitic and tuberculous affections of the joints. *Syphilitic* arthritis is usually accompanied by other syphilitic symptoms, especially keratitis, and ordinarily yields to antisyphilitic treatment. The differentiation between simple chronic arthritis and *tuberculous* joints is quite difficult, since, as previously mentioned, the latter may follow upon the former. However, the absence of temperature and failure to obtain a positive tuberculin reaction speak in favor of chronic non-tuberculous arthritis. The finding of a tuberculous exudation in the affected joint, of course, is decisive in the diagnosis.

Persistence in treatment.

As the prognosis in protracted cases is very bad, active treatment should be begun with early and not too rapidly discontinued in disgust because of more or less persistent failure to effect a cure. The salicylates with small doses of sodium iodid internally and pure ichthyol externally should be given a thorough trial. Where stiffness and swelling of the joints prevail, daily gentle massage preceded by a hot local bath and followed by hot, moist compresses often works wonders. Passive motion should be practised early and where the contractures are very pronounced one should not hesitate to reduce the same under anesthesia and proceed with the treatment just outlined. Concomitant acute symptoms should be treated in the same manner as in acute rheumatism, and when there is reason to believe that the diseased condition is the result of faulty metabolism (intestinal intoxication or uric acid diathesis?) the dietary should be regulated accordingly (exclusion of meats, acids, liquors, etc.).

Strict diet.

℞ Natrii iodidi	gr. xv	1.0
Ext. hyosciami fl.	gtt. vj	0.4
Natrii salicyl.	ʒj	4.0
Syr. sarsaparillæ comp.	℥j	30.0
Aq. destil. q. s. ad	f℥iij	90.0

M. Sig.: ʒij every four hours for a child 6 years old.

STILL'S DISEASE.

This affection generally sets in during the first three or four years of life, and attacks girls more frequently than boys. It is characterized by gradually developing stiffness and enlargement of several joints, beginning with the knee, wrists and cervical vertebræ, and gradually extending to the fingers and toes. It differs pathologically from rheumatoid arthritis or tuberculosis in that it is free from destructive or proliferating processes of the bony structures. The enlargement of the joints is due purely to thickening of the soft tissues. Aside from the articular involvement Still's disease is characterized by a more or less marked enlargement of the lymphatic glands (axillary, cervical and mesenteric) and of the spleen. It is occasionally associated with a slight rise of temperature, and shows a tendency to pericardial and pleural adhesions.

Gradual stiffness and enlargement of joints.

Lymphadenitis.

It is a very chronic, incurable affection of unknown etiology. Its progress may be partially arrested by the therapeutic measures outlined under "chronic rheumatism" (*q. v.*).

RHEUMATISMUS NODOSUS INFANTILIS.
ERYTHEMA NODOSUM.
PELIOSIS RHEUMATICA (PURPURA RHEUMATICA).

These three distinct diseased conditions are grouped together to facilitate their identification. They have several symptoms in common, and bear a close resemblance to rheumatism. Their true nature, however, is a matter of conjecture, and with our present ignorance as to the identity of the specific rheumatic germ there are no means of corroboration or of contradiction of any of the numerous assumptions advanced by different authorities.

Their correlation to rheumatism.

1. RHEUMATISMUS NODOSUS INFANTILIS.

It is peculiar of early childhood and occasionally follows a protracted or recurrent attack of rheumatism, especially in association with grave cardiac manifestations. It is characterized by the (often symmetrical) appearance, chiefly about the joints and the tendon insertions, of several nodules (*noduli* or *osteomata rheumatici*) which grow to a perceptible size, and then either undergo regressive, fatty metamorphosis and absorption, or persist, become calcified and acquire a bony consistence. The nodules (*exostoses*) vary in size from a small pea to a plum, and in number from one to a hundred. They are at first soft, flat and painful or tender to the touch, and later they become harder and rounder, resembling the fibromatous and osteomatous growths observed in "myositis ossificans" and in "multiple exostoses" (*q. v.*). Treatment, antirheumatic.

Nodules near joints.

2. ERYTHEMA NODOSUM.

Until recently this affection has been looked upon as a skin disease pure and simple. The sudden appearance, the rise of temperature, the self-limited course, and its association with more or less marked constitutional symptoms and occasionally grave complications (principally rheumatic pain, bleeding from mucous membranes and heart trouble) stamp it, however, as an acute infectious disease of obscure etiology. Locally it is characterized by the appearance, chiefly on the anterior portion of the lower legs and forearms, of from a pea- to a walnut-sized, pale-red painful nodules which at first resemble contusions (*erythema contusiforme*). They gradually disappear, changing in color to bluish, green and yellow within from two to three weeks, as a rule, without any specific medication. Complications of the heart and joints demand antirheumatic treatment.

Pale-red nodules in front of lower legs and forearms.

3. PELIOSIS (PURPURA) RHEUMATICA
(Schoenlein's Disease).

The local manifestations of this affection consist of variously sized bright- to bluish-red hemorrhagic spots which are uninfluenced by pressure with the finger. Here and there they present a central papular hardness. The eruption is usually limited to the lower extremities, especially about the knees and ankles, but the upper extremities may be affected as well. The appearance of the eruption is preceded and accompanied by articular pain and swelling, occasionally soreness of the soles of the feet, and difficulty in walking. Fever and constitutional symptoms are ordinarily slight.

Hemorrhagic spots about knees and ankles.

The prognosis is usually favorable, but the disease manifests a tendency to recurrences, and to cardiac complications. Treatment, symptomatic (salicylates; hemostatics, such as iron, gelatin, turpentine; rest).

Tendency to heart disease.

MYOSITIS

(Inflammation of the Muscles).

The causes of myositis are very numerous. We had occasion to refer to scarlatinal and rheumatic myositis. It may also be traumatic, gonorrheal, syphilitic and tuberculous in nature, and is occasionally observed in connection with other infectious disease, *e.g.*, typhoid. Myositis is characterized by pain, swelling and loss of function of the affected muscles, and in protracted cases by contractures. Where pain predominates and the swelling is slight, myositis may readily lead to diagnostic errors—as emphasized in the discussion of "muscular rheumatism" (see page 417). Traumatic, syphilitic and tuberculous myositides are prone to lead to suppuration, while simple so-called rheumatic myositis eventually subsides either spontaneously or under antirheumatic treatment.

In connection with divers microbic affections.

Tendency to suppuration.

POLYMYOSITIS.

This form of general myositis is of much graver nature than the aforementioned varieties. It occurs either primarily, without any apparent cause, or secondarily as a result of parasitic infection, such as trichinæ, echinococci, cysticerci, etc.

Due to trichinosis, echinococci, cysticerci, etc.

Preceded by prodromata of a few days' duration, consisting of headache, muscular pain, anorexia and slight fever, the condition rapidly grows worse; the temperature rises, and edema of the eyelids and face appears which soon spreads over the entire surface of the body. Beginning also with the face, the entire musculature of the body (least marked in the hands and feet) rapidly becomes stiff, board-like, and very painful, so much so that the different functions of the body (mastication, deglutition, respiration, etc.) are interfered with and the condition greatly resembles that of cerebral rigidity.

Stiffness interfering with different functions of the body.

In some cases cutaneous edema predominates (*dermatomyositis*), in others a hemorrhagic condition of the skin and mucous membrane (*polymyositis hemorrhagica*). Some cases develop very slowly and lead to overgrowth of the connective tissue (*myositis fibrosa*). In trichinosis the polymyositis is usually

Trichinosis associated with gastro-intestinal disturbance.

preceded by gastrointestinal disturbances, and the stools and the muscles reveal trichinæ spiralis.

In children the course of the disease is usually milder than in adults and, as a rule, ends in recovery.

Treatment: symptomatic; thorough cleansing of the alimentary tract; relief of pain by antispasmodics.

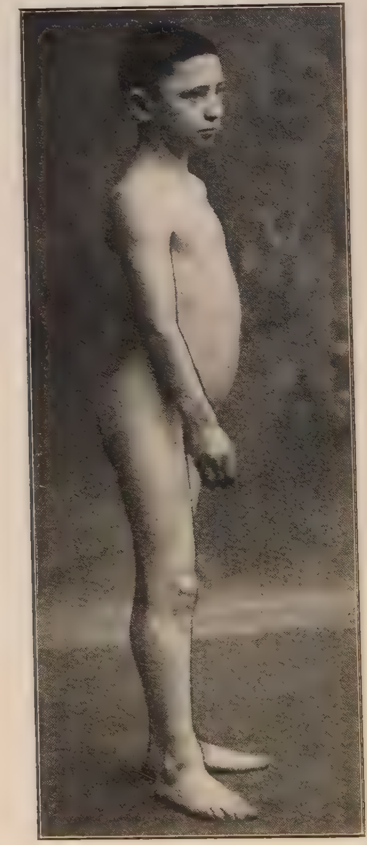

Fig. 135.—Multiple Exostoses. The tumors, varying in size from a pea to a walnut, were especially numerous at the costosternal articulations, the wrist-, knee- and ankle-joints. (Steffen.)

MYOSITIS OSSIFICANS.

Myositis ossificans multiplex progressiva is a ~~disease of~~ childhood, the majority of the cases on record having been observed in children under ten years of age. Anatomically it is characterized by progressive interstitial connective-tissue proliferation, with consecutive ossification. The affection begins with the muscles of the neck and back, then spreads to those of the extremities, and, finally, involves the masseter and temporal muscles.

Begins in the neck and back.

The etiology of the disease is unknown. It is possibly due to a congenital anomaly of the connective-tissue structure.

The onset is sudden with fever, and a soft, painful swelling of a section of a muscle, over which the skin appears reddened and edematous.

Fever; localized swelling, gradually becoming of bony consistence.

The febrile symptoms soon abate, but the swelling in the muscle persists, and gradually—it sometimes takes years—assumes a bony consistence. Several muscles may thus become affected, leading to disturbances of motion, rigidity and deformities, and ossification of a large portion of the body so that the patient becomes bedridden for life. The prognosis, therefore, is grave, and life is endangered early if the muscles of mastication and respiration are involved.

Treatment.—Avoidance of traumatism; the iodids internally and externally; gentle massage and hot baths.

MULTIPLE EXOSTOSES.

Bone tumors in children may be congenital or acquired. The latter variety has been spoken of in connection with rheumatism (see page 422). Congenital exostoses may escape observation for several years and then erroneously be attributed to acquired causes. The etiology of congenital exostoses is obscure. Some cases are traceable to syphilis hereditaria. Bone tumors localized in the immediate neighborhood of joints and interfering with motility should be extirpated.

Congenital and acquired.

POLIOMYELITIS ACUTA.

(See page 529.)

CHOREA ACUTA.

(See page 563.)

CHAPTER X.

Diseases of the Heart.

CONGENITAL HEART DISEASE

[illegible]

[illegible] heart disease are very delicate. [illegible] born asphyxiated and if resuscitated remain [illegible] atelectatic, cry feebly, breathe super-[illegible] a very weak pulse and [illegible] born prematurely [illegible] of other parts of the body. Some [illegible] appearance of the fingers and toes [illegible] them later. If they survive for any [illegible] growth and development are very much [illegible] begin to hold up the head or sit up [illegible] the normal baby. When they start to [illegible]. They rarely creep and when on the [illegible] to lift themselves. They are very [illegible] once taken sick they are very slow to [illegible] to gastrointes-[illegible] comparatively simple nature. If they live [illegible] more frequently exposed to acute con-[illegible] diseases their weakened constitution forms [illegible] the contraction of these affections, and is [illegible] them.

[illegible] children with congenital heart [illegible] few years. Death sometimes occurs [illegible] the course of other diseases which in [illegible] dangerous to life, especially respiratory [illegible] heart defect is very mild in nature, chil-[illegible] very rarely survive the age of puberty.

[illegible] cyanosis, or "blue sick-[illegible] symptom of congenital heart [illegible] of the fingers and toes [illegible] diagnostic importance has been greatly exaggerated. [illegible] congenital vitium [illegible]

[illegible] of the Newly Born."

The course of congenital heart disease varies, of course, with the severity of the defect, but practically resembles that of acquired vitium cordis, which is fully described in other parts of this treatise. The following are the most common congenital heart affections:—

Resembles acquired heart disease.

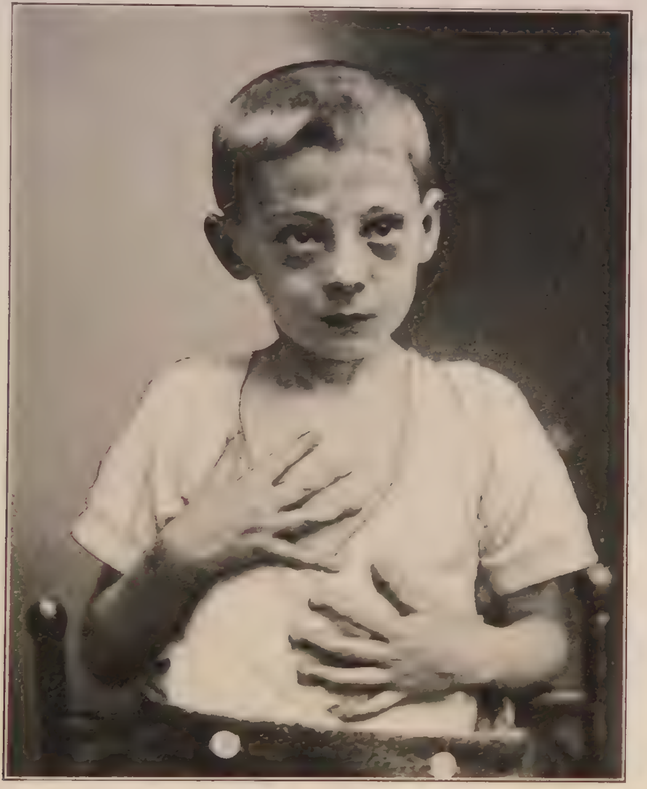

Fig. 136.—Vitium Cordis. "Morbus Cœruleus." Note "club-shaped" fingers and cyanosis (represented by dark patches on face and lips), in a child 8 years old. (*Sheffield.*)

PERSISTENCE OF THE FORAMEN OVALE.

This condition is the result either of faulty construction of the foramen or its valves, or defects in other portions of the heart (*e.g.,* stenosis of the pulmonary artery) which by indirect blood-pressure prevent complete obliteration of the foramen.

It is the most frequent kind of congenital heart disease, but is not always recognizable during life. In the presence of clinical symptoms the diagnosis may be based upon predominance of

Most common.

Systolic basic murmur.

cyanosis, a systolic blowing sound at the base of the heart or over the third or fourth costal cartilage.

PERSISTENCE OF THE DUCTUS ARTERIOSUS BOTALLI.

Hypertrophy of right ventricle.

Complete obliteration of this duct is supposed to occur by the end of the third month. This may be retarded or may entirely fail—usually in cases where the left ventricle is not properly filled with each heart-cycle (*e.g.,* in atelectasis, fetal pneumonia, stenosis of the pulmonary artery)—in which event the blood from the pulmonary artery continues to flow through the ductus arteriosus to the insufficiently filled aorta. As a result of this anomaly there develops sooner or later hypertrophy of the right ventricle, usually with dilatation of the pulmonary artery.

The symptomatology is very variable.

Thrill: systolic murmur over precordium.

In cases of only partial patency the symptoms may be so slight as to escape observation. Complete patency of the duct very gradually gives rise to the following group of symptoms: Disposition to respiratory affections, cyanosis, or waxy pallor; dyspnea, cool extremities, palpitation, a thrill over the anterior chest wall, increased cardiac dullness to the right, accentuation of the second pulmonic sound, loud systolic murmur over the precordium, often epistaxis or hemorrhage from other mucous membranes; finally, sometimes not until after several years of existence, marked symptoms of failure of compensation with rapid fatal termination.

DEFECTS IN THE SEPTUM VENTRICULORUM
(Communication of the Ventricles).

It is a very common condition, most frequently the result of fetal myocardial diseased processes, and not rarely coexisting with congenital stenosis of the pulmonary artery. The defect is situated either in the anterior or posterior portion of the septum. Very rarely the whole wall between the ventricles and auricles is absent so that all four heart cavities communicate.

Marked cyanosis soon after birth.

Accentuation of the second pulmonic sound; overfilling of the veins; marked cyanosis developing soon after birth or, more gradually, some time after, and hypertrophy and dilatation of the right ventricle—all point to a defect of the ventricular septum. A positive diagnosis, however, is almost impossible during life of the patient.

The prognosis is very bad.

X

CONGENITAL STENOSIS OF THE PULMONARY ARTERY.

The stenosis may involve the orifice alone, the entire trunk, or the branches of the pulmonary artery. Accordingly the symptomatology varies with extent and location of the lesion. As a rule, there is marked cyanosis from birth. Some children are born asphyxiated, and if resuscitated continue to suffer from attacks of suffocation and convulsions, to which they usually succumb within the first few days of life. Stronger children may survive these attacks, gain some strength, lose part of the cyanosis and live several years.

Born with cyanosis.

Physical examination reveals arching of the anterior left chest wall; enlargement of the cardiac area, chiefly to the right; diffuse systolic murmur, heard loudest over the left second and third costal cartilages, and often a purring thrill on palpation.

Basic systolic murmur.

CONGENITAL STENOSIS OF THE TRICUSPID VALVE.

It is usually the result of an anomalous or excessive development of muscle substance instead of the valve, or of fetal endocarditis, and is often associated with other congenital heart defects.

The symptomatology resembles that of stenosis of the pulmonary artery, except that the murmur is heard loudest over the fourth and fifth costal cartilages, and hypertrophy of the right side is either absent or very slight.

Murmur over tricuspid valve.

The prognosis is unfavorable.

CONGENITAL STENOSIS OF THE OSTIUM ATRIO-VENTRICULARE SINISTRUM

(Stenosis of the Aorta).

The stenosis may be situated at the point of origin of the aorta; at any place throughout the entire aortic system; or at the ductus Botalli.

As a result of either one of the aforementioned conditions there is hypertrophy of the left heart. Varying with the seat of the atresia, the blood-vessels given off above the lesion may be abnormally filled with blood, while those emerging below the lesion suffer from a deficiency of it. Between the two groups of vessels a collateral circulation is usually established, which may frequently be recognized by numerous, visible, actively pulsating, subcutaneous blood-vessels over the thorax. A systolic murmur is often heard over the dilated arteries. The heart is usually

Hypertrophy of left heart.

Pulsation of blood-vessels.

Arterial murmur.

free from any auscultatory signs, unless the orifice of the aorta be involved, when a loud systolic murmur may be heard at midsternum.

The patient may live for several years—until compensation ruptures. Death sometimes ensues very suddenly from rupture of a group of vessels above the stenosis.

Treatment.—The treatment of congenital heart diseases is essentially the same as that of acquired, and is fully outlined on page 443. Complete rest in the strictest sense of the word will help to prolong life—possibly to an advanced age.

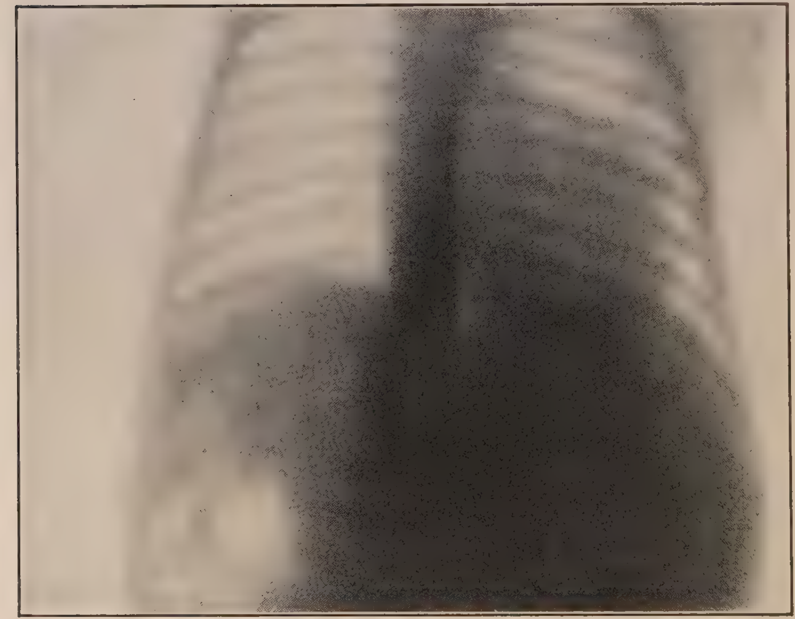

Fig. 137.—Dextrocardia in a Girl 6 Years Old (skiagram; back view). (*Sheffield.*)

Complete rest.

DEXTROCARDIA.

Among the few congenital malpositions of the heart (*mesocardia*—the heart occupies a *central* position of the chest-wall; *ectopia cordis*—the heart may be situated either between a fissure in the sternum immediately beneath the skin, in the neck, or in the abdomen below the diaphragm) dextrocardia, or transposition of the heart to the right side, is of special interest inasmuch as it very rarely interferes with the life or welfare of the patient.

Dextrocardia is often associated with a general transposition of the viscera. The aorta and its branches usually remain in their normal situation. Dextrocardia should not be confounded with displacement of the heart by large effusions or growths in the thoracic cavity.

Often transposition of viscera.

ACQUIRED HEART DISEASE.

MYOCARDITIS.

Inflammation of the muscular tissue of the heart is occasionally congenital, a sequel of infection during fetal life, but most frequently acquired, occurring either secondarily to acute infectious diseases, or as a result of extension of an inflammation from the inner or outer lining of the heart.

Usually acquired.

The inflammation may be diffuse or circumscribed, and as in adults either plastic or *interstitial,* or degenerative or *parenchymatous.*

The interstitial variety of myocarditis usually leads to suppuration and abscess formation of the musculature. In parenchymatous myocarditis the transverse striæ of the fibrillæ appear lost, the muscle consisting chiefly of fatty and granular matter.

The course of the disease varies greatly with the underlying cause and the rapidity of the inflammatory process.

Interstitial.

In the majority of instances *interstitial* myocarditis is complicated by endocarditis and pericarditis, and in consequence of preponderance of the signs of the latter affections it is very seldom possible early to diagnose the existence of the myocarditis. In cases where the inflammation is circumscribed, myocarditis may be surmised by the sudden precordial pain, dyspnea, high fever, restlessness and delirium. The apex-beat and pulse are weak, arhythmic and rapid. Death is the usual termination; not rarely occurring suddenly (sometimes from rupture of the abscess in the heart cavities) with symptoms of sudden collapse.

Sudden precordial pain.

Arhythmia.

Parenchymatous.

Slow course.

Parenchymatous myocarditis ordinarily runs a slow and latent course. Occasionally, however, the degenerative process develops quite rapidly. Extreme pallor, breathlessness, and weak and galloping pulse point to the involvement of the myocardium, but in the early stages the diagnosis can rarely be made with any degree of certainty. As the disease advances and symptoms of cardiac dilatation and passive pulmonary congestion set in, the diagnosis is fairly certain.

Galloping pulse.

The treatment is the same as in endocarditis (*q. v.*).

PERICARDITIS.

Like pleuritis, inflammation of the pericardium may occur in dry form or with an effusion. The exudation may be serofibrinous, hemorrhagic, or purulent. Dry as well as exudative pericarditis usually gives rise to inflammatory adhesions between the pericardium and heart, and occasionally to the anterior and posterior chest-walls and vertebral column. Chronic pericarditis is productive of grave disturbances of the circulation, cardiac hypertrophy and dilatation.

Inflammatory adhesions.

The gravity of this affection should, therefore, not be underestimated. The prognosis is serious, especially in the secondary variety occurring in connection with tuberculosis, septic processes, pleuropneumonia, caries of ribs or vertebræ, severe exanthematous diseases (*e.g.*, scarlatina), purpura hemorrhagica, chronic nephritis, etc. It is less dangerous in primary, usually rheumatic form, particularly if the patient is over three years of age, or when caused by syphilis and is detected and treated early.

Bearing in mind the etiologic factors just enumerated, we can readily appreciate that pericarditis in children must be quite common. Indeed, there is ample reason for the belief that in children over three years of age pericarditis is more frequent than endocarditis—with which affection, by the way, it is not rarely associated.

The onset of primary pericarditis is usually very sudden, but sometimes, like in the secondary variety, it may be insidious. Ordinarily it is ushered in with high temperature, vomiting, cardiac oppression, dyspnea, and accelerated pulse. Cough is an early symptom and, in the presence of an effusion, quite pronounced. This symptom is probably due to cardiac pressure against the lungs. The pulse, which in dry pericarditis is strong, in marked exudative pericarditis it is often very feeble, barely perceptible, and irregular. Pain is frequently intense, especially if associated with polyarthritis. The patient is restless, sleepless; his expression of the face is anxious, denoting great suffering. Of course, the symptomatology is greatly modified by that of the underlying affection, if existing.

Fever; cardiac oppression; cough.

The physical signs vary with the stage of the disease. Before the development of the effusion auscultation elicits superficial, exocardial, to-and-fro friction and creaking sounds, limited over the cardiac region, often changeable with position of the patient

To-and-fro friction.

and audible independently of the heart sounds. Endocardial murmurs may coexist. When serous effusion occurs, the friction sound is found diminished or absent, the heart impulse very feeble (whereas the pulse may be felt quite strong), the area of heart-dullness greatly increased and wider at the apex than at the base, and when the effusion is large we notice also diminution of the respiratory movements of the left side. With absorption of the fluid in the pericardium there is gradual return of the symptoms of the first stage and in favorable cases restitutio ad integrum, or, quite frequently, supervention of pericardial adhesions with consecutive systolic retraction of the chest-wall over the entire precordium. The percussion symptoms are not absolutely reliable, since increase of the area of cardiac dullness is also observed in dilatation or hypertrophy. There are, however, several other distinctive features which render the differentiation of pericardial effusion from enlarged heart possible. Thus, in dilatation or hypertrophy of the left ventricle the apex-beat is felt at the extreme left limit of the dullness and at its lowest level, while in effusion the apex-beat or rather the heart-impulse is at a spot inside and above the boundaries of the cardiac dullness, somewhere between the fourth ~~or~~ third interspace. In pericarditis the dullness develops much more acutely than in an enlarged heart, which latter occurs usually secondarily to more or less chronic valvular disease. However, we should bear in mind that pericarditis, acute or chronic endocarditis, and hypertrophy and dilatation may coexist and give rise to a symptom-complex beyond the possibility of individualization. For the differentiation between pericarditis and endocarditis the reader is referred to the discussion of the latter affection (see page 439).

Feeble impulse.

Wide apical dullness.

Differentiation from hypertrophy.

The nature of the effusion can readily be ascertained by exploratory puncture, but even without it we may surmise the presence of *pus* if the pericarditis develops secondarily to septic processes; *blood*, after severe trauma, and *serum*, in primary, usually rheumatic, pericarditis.

Nature of effusion.

The determination of the character of the effusion is important especially as regards the further course and treatment of the disease.

Rheumatic ~~pericarditis~~, if free from complications, usually lasts for from two to three weeks or longer. After about ten days there is gradual evanescence of the symptoms. Not infrequently, however, the "apparent" recovery is only temporary, inasmuch as there may be a return of the effusion, or development of valvular deposits, which sooner or later give rise to marked valvular disease. These manifestations are particularly prone to occur in pericarditis with polyarthritis. Pericarditis, like endocarditis, not rarely precedes the joint symptoms, may run a latent course and disappear again without being detected—possibly not until repeated recurrences and appearance of complications. More rarely, pericarditis ends in death either rapidly as result of cardiac muscular insufficiency and pulmonary edema, or slowly from early complications, such as pleurisy, pneumonia, severe adhesions, endocarditis, etc.

Rheumatic.

Purulent.

Purulent pericarditis pursues a much more violent course. Extreme fatigue, severe attacks of syncope and pyemic fever predominate, while the local symptoms are comparatively insignificant. Even the exudation is often slight. When it occurs in conjunction with tuberculosis, it is very malignant in character. It is then manifested by enormous hypertrophy of the pericardium, extensive adhesions, large quantity of pus between the heart and pericardium, and numerous tubercles in the latter. It is invariably fatal. The same holds true for pyemic pericarditis, in which streptococci, pneumococci, staphylococci and less frequently gonococci act as the principal exciting cause.

Tuberculous.

A disease presenting so many phases as pericarditis can at best be treated only symptomatically. Absolute rest in bed, an icebag, or a flaxseed-meal poultice, to the precordium, and sodium salicylate (1 grain for every year of the child's age every two hours) and codein ($\frac{1}{100}$ of a grain every six hours) internally will often do well in rheumatic cases. In large pericardial serous effusions with threatening syncope we may try free diuresis, with or without aspiration (in the fifth intercostal space a little to the left of the border of the sternum). The latter procedure frequently proves useful in small non-tuberculous purulent effusions, while in large purulent effusions incision and drainage are preferable to aspiration.

Rest; icebag; salicylates.

Aspiration.

In quite a number of cases sodium iodid seems to exert a specific effect, and bearing in mind also the possibility of underlying latent syphilis we should always administer this remedy irrespective of the variety of the pericarditis and the mode of treatment simultaneously employed. Digitalis or strophanthus may be given to strengthen the heart.

Iodids.

ENDOCARDITIS ACUTA.

Rheumatic.

Streptococcic, etc.

The etiologic factors of acute endocarditis are essentially the same as in pericarditis (*q. v.*), except that the former is more frequently associated with rheumatic affections, such as arthritis, chorea, tonsillitis, erythema nodosum, etc., and not rarely complicates pericarditis. Invasion of the endocardium by the streptococcus, staphylococcus, pneumococcus, the bacillus pyocyaneus, tubercle bacillus and gonococcus usually occurs through the circulating blood, giving rise to a pathologic condition very similar to that observed in adults.

Pathologic findings.

The inflammation which is usually limited to the left side of the heart first attacks the vascular layer of the endocardium between the muscular and fibrous coats, resulting in an exudation of lymph and serum principally beneath and on the free surface of the membrane covering the valves and chordæ tendineæ. As the disease progresses large or small papillary nodules, vegetations, are formed on the endocardium—*endocarditis verrucosa,* or ulcerations occur as a result of destruction of the superficially necrosed tissue—*endocarditis ulcerosa.* The latter condition is usually found in the malignant, usually septic, form of endocarditis. During the course of endocarditis many organs of the body, *e.g.*, the kidneys, spleen, brain, etc., may become implicated through emboli composed of masses of fibrin or necrosed tissue which become detached by the circulating blood principally from the irregular valvular deposits. In septic cases these emboli give rise to abscesses. It is well to remember, however, that moderately severe cases of endocarditis may go on to complete recovery, and leave no trace of the original inflammation on the endocardium; furthermore, that slight valvular vegetations are not infrequently found post mortem without any apparent clinical signs of heart disease during life.

Chills, fever, precordial distress and cough.

This latter observation can readily be explained by the fact that mild endocarditis is not rarely masked by the course of another disease, and unless presenting marked disturbance of the circulation is very apt to be overlooked. More often, of course, endocarditis sets in with severe unmistakable symptoms. The patient vomits, suffers from chills, more or less high fever (102° to 105°), precordial distress, short cough, dyspnea, and accelerated and sometimes irregular pulse. These symptoms, however, are not sufficiently characteristic of endocarditis and may still

Usually mitral murmur.

leave the nature of the disease obscure until the subsequent appearance of local signs, especially of a systolic heart murmur, audible chiefly at the apex (the mitral valve being most frequently involved) or also over the whole cardiac region. As will be seen later (see "Endocarditis Chronica," page 439) murmurs may subsequently develop at the various orifices of the heart, and at a later stage of the disease additional physical signs (dilatation or hypertrophy) may be obtained by percussion.

Occasionally (in children less frequently than in adults) acute endocarditis pursues a very septic and often violent course—*endocarditis maligna* (ulcerosa). It may be preceded by pneumonia, exanthematous diseases, septic processes in some other parts of the body, etc., or occur without any apparent cause and exhibit a symptom-complex resembling either a low typhoid state or cardiac insufficiency with acute dilatation (cyanosis), and loud murmurs at the various orifices. The duration of malignant endocarditis varies. Ordinarily it runs a protracted course with irregular temperature, chills, rigors and sweats. Sooner or later emboli develop in different organs of the body and the capillaries of the skin the superadded symptoms varying, of course, with the organ affected. If the brain is involved, we find palsies with disorder of consciousness; if the spleen, enlargement of this

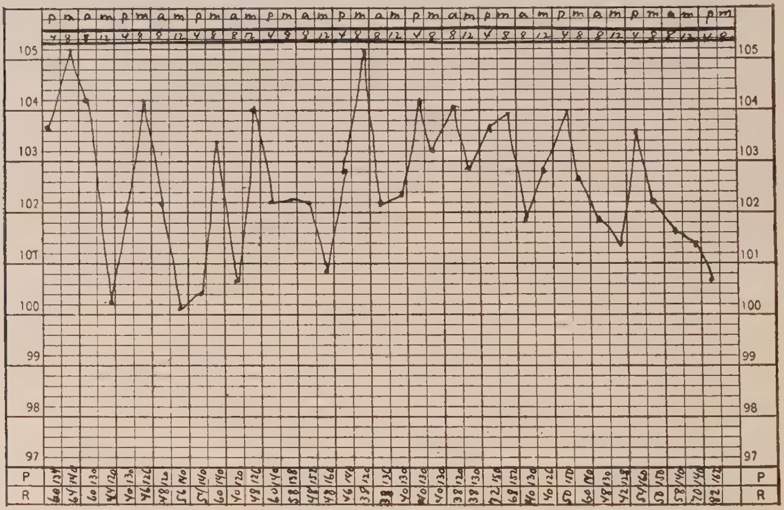

Fig. 138.—Fever Curve of Malignant Endocarditis in a Child 3 Years Old. (*Sheffield.*)

Malignant form.

Septic symptoms.

Complications.

organ and tenderness; if the kidneys, albuminuria, hematuria and anasarca; if the skin, petechiæ and a pustular eruption. It is not rarely complicated also by purulent pericarditis. When malignant endocarditis runs so very violent a course it, as a rule, terminates fatally within a few days. On the other hand, simple, benign endocarditis in children is usually not dangerous to life. If free from complications the symptoms begin to subside after about a week or ten days, eventually leading to recovery in about four weeks. In quite a number of cases, however, it is followed by permanent valvular disease, with or without cardiac hypertrophy (see "Endocarditis Chronica"). Death is usually due to cardiac paralysis.

Benign endocarditis may be mistaken for *dry* pericarditis, especially if the former is associated with articular rheumatism. The following table contrasts the most important distinguishing features. Both diseases, however, may coexist.

Differential diagnosis from pericarditis, typhoid, malaria, miliary tuberculosis and pyemia.

SIMPLE ENDOCARDITIS.	DRY PERICARDITIS.
Blowing or musical sound.	"To and fro" friction or creaking sound.
Sound is associated with systole or diastole.	Not necessarily. May be heard at any period of cycle.
Sound is distant.	Near to the ear.
Sound is uninfluenced by pressure with the stethoscope.	Increased.
Sound is conducted upward, to the axilla, and to the back.	Not so.
Sound usually loudest at apex.	Anywhere over precordium.

The diagnosis of ulcerative endocarditis is very difficult, especially in the incipient stage, before the appearance of a heart murmur. Whenever several orifices are the seat of the murmur, paroxysms of cyanosis and dyspnea and irregular temperature predominate, and cardiac dullness is increased, the diagnosis of malignant endocarditis is justified. The elimination of typhoid, irregular malarial fever, miliary tuberculosis, and pyemia, the four affections with which malignant endocarditis is most apt to be confounded, will greatly facilitate the diagnosis.

The treatment of endocarditis is essentially the same as in pericarditis—purely symptomatic. (See "Pericarditis," page 436.)

ENDOCARDITIS CHRONICA
(Valvular Heart Disease).

Chronic endocarditis ~~is most~~ frequently a sequel of acute inflammation of the endocardium, especially of the valves, and pathologically consists of proliferation and thickening of the

Limited to left heart.

valvular connective tissue with a great tendency to contraction and adhesions, and very rarely to calcification. The chronic inflammatory process is usually limited to the left side of the heart except in cases developing during fetal life, when the reverse is the case.

Hypertrophy.

Dilatation.

Coincident with the inflammatory process in the endocardium, the cardiac musculature undergoes gradual enlargement. This hypertrophy, unless assuming exceptionally large dimensions (*e.g., cor bovinum*), is strictly speaking not a disease *per se,* but, on the contrary, an effort on the part of nature to overcome or undo the evil effects of the disease. As the disease advances and the heart muscles lose their power, get exhausted, the hypertrophy is replaced by dilatation, indicating that compensation has "ruptured," and that disease is beyond control.

Anemia.

Pulmonary edema.

Until failure of compensation has occurred children may for years remain apparently free from any marked disturbances of health, except, perhaps, rapid fatigue, palpitation of the heart on exertion, progressive anemia and malnutrition notwithstanding good appetite and digestion. Indeed, it is often chiefly for disturbance of the latter that the patients are brought to the physician. Shortness of breath, which increases on exertion, usually forms an early manifestation of failing compensation. It is the result of stasis in the pulmonary circulation with consecutive impairment of aëration. This sooner or later leads to passive congestion of the pulmonary alveoli, giving rise to bronchitis, with an irritable cough, and, as the heart-failure increases, to paroxysmal attacks of dyspnea especially at night ("cardiac asthma"), pulmonary edema, cyanosis, and occasionally to hemorrhagic infarcts in the lung with consecutive hæmoptœ.

Complications.

Simultaneously with the aforementioned manifestations, pathologic changes go on also in other internal organs—the liver, spleen, and kidneys. The liver and spleen are enlarged, and by pressure upon neighboring thoracic organs increase the dyspnea. As a sequel of the passive congestion of the liver and stasis in the blood-vessels of the stomach and intestines, numerous gastro-intestinal disturbances—*e.g.*, anorexia, vomiting, constipation—develop, which add misery to the patient's painful existence.

The changes in the kidneys are manifested by diminution in the quantity of urine, often albuminuria (slight), hyaline and cylindrical casts, and occasionally white and red blood-corpuscles —signs of passive congestion.

Dropsy.

Remissions.

Heart-failure.

With increasing venous stasis there is coincident transudation of the fluid of the blood from the capillaries into the meshes of the tissues, leading to edema. At first the dropsy is limited usually to the ankles and eyelids, but as the disturbance of circulation advances it grows worse and involves the entire integument and the internal cavities, especially the abdominal and pleural cavities. Notwithstanding the extreme gravity of the condition, the end is not always as near as would be expected. The inherent power of the infantile heart is still capable of temporary reparation. The arhythmia, dyspnea, and dropsy may disappear; the appetite and nutrition may improve; the tottering patient may again be up and around; in fact, may appear at his best. Exacerbations and improvements of the disease may come on several times. The improvement is but short-lived. Very soon the symptoms return, and, as a rule, with greater severity. Finally, after a more or less prolonged period of illness the patient succumbs to heart-failure. Occasionally death occurs suddenly after severe exertion. Quite a number of children are carried away by intercurrent infectious diseases, pericarditis or recurrent endocarditis. The physician should therefore always be very guarded in the prognosis. The relative gravity of valvular lesions is as follows: Tricuspid regurgitation; mitral regurgitation; mitral stenosis; aortic regurgitation; pulmonic stenosis; aortic stenosis.

Relative gravity of lesions.

Differential Diagnosis.[1]—As the physical signs of valvular heart disease in children differ but little from those in adults, we will review only the most essential differential points of diagnosis.

MITRAL REGURGITATION.

Systolic murmur at apex.

Insufficiency of the mitral valve is characterized by a systolic blowing murmur which is loudest at the apex and transmitted to the axilla and near the lower angle of left scapula. Accentuation of the second pulmonic sound. Hypertrophy of the left ventricle, and later left auricle, and sequential hypertrophy of the right ventricle. The pulse may be normal or accelerated, and with disturbed compensation—which may not occur for many years—irregular and unequal.

MITRAL OBSTRUCTION.

Presystolic or diastolic murmur at apex.

It is frequently associated with insufficiency. The murmur is usually presystolic or also diastolic, best heard at the apex; may be conveyed to the fourth interspace, but never to the angle of the scapula. The pulmonic second sound is accentuated and sometimes double. It frequently leads to hypertrophy of the left auricle and right ventricle.

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[1] See Fig. 16.

AORTIC REGURGITATION.

Diastolic murmur at right base.

Aortic insufficiency is rare in children. It is accompanied by hypertrophy of the left ventricle, and often pulsation of the arteries of the neck. The murmur is diastolic, loudest at the insertion of the right second costal cartilage and over the upper portion of the sternum. At first the murmur is quite noisy, but with ensuing disturbance of compensation it loses its intensity. It is usually combined with aortic stenosis, becoming the gravest form of valvular disease of childhood. It sometimes causes sudden death, and but few children survive the age of puberty. Aortic regurgitation may often be recognized by its peculiarly collapsing pulse—the water-hammer or Corrigan's pulse.

AORTIC OBSTRUCTION.

Systolic murmur at right base.

This affection is usually observed in older children in connection with aortic insufficiency. The murmur is harsh, systolic, heard loudest over the aortic orifice, transmitted to the right, and sometimes over whole length of sternum, and the arteries of the neck. Hypertrophy of the left ventricle.

TRICUSPID REGURGITATION.

Systolic murmur at xiphoid.

Except as a congenital defect it most frequently occurs secondarily to affections of the left heart. Auscultation reveals a systolic, blowing murmur heard loudest over the lower part of the sternum (xiphoid) and at the juncture of the fourth costal cartilage. Second sound is weak. Jugular pulsation. Hypertrophy and dilatation of the right heart. In severe cases cyanosis, and pulsation of the liver.

TRICUSPID OBSTRUCTION.

This condition is extremely rare; hence, calls for no detailed discussion. No particular change in size of the heart is known. (See "Congenital Heart Disease.")

PULMONIC REGURGITATION.

Insufficiency of the pulmonic valve is chiefly congenital, rarely acquired. The murmur is diastolic and limited to the site of the valve—at the junction of the left second costal cartilage and the sternum. Unlike that of aortic insufficiency it is not transmitted to the arteries of the neck. Hypertrophy of the right heart.

Diastolic murmur at left base.

PULMONIC OBSTRUCTION.

Principally a congenital malady. The murmur is basic, systolic, heard loudest at the left second costosternal junction. It is associated with hypertrophy of the right ventricle, and sometimes cyanosis. (See "Congenital Heart Disease.")

Systolic murmur at left base.

Functional or inorganic murmurs—those arising during the course of acute febrile diseases or in association with anemic conditions—may be mistaken for organic murmurs of valvular heart disease. The chief points of distinction between them are as follows: Functional murmurs are inconstant, heard principally at the base with systole, not transmitted away from the heart, and usually disappear with defervescence, or improvement of the anemia. Functional murmurs are very rare in children up to seven years of age.

Differentiation from organic murmurs.

The management of chronic valvular heart disease in children is the same as in adults. It differs with the stages of the disease—when compensation is intact, and when it "ruptures."

STAGE OF COMPENSATION.

The well-being and longevity of the patient stand in direct ratio to the capacity of the heart to compensate its insufficiency by secondary hypertrophy of the musculature of one or more of its chambers. Hence, the aim in the treatment of chronic valvular heart disease should be directed chiefly to the maintenance of compensatory hypertrophy. Bearing in mind the facts that with increasing circulatory disturbance there is on the part of the heart a spontaneous muscular development to overcome its difficulties as long as its supply of nourishment is sufficient, and its hypertrophic process is not interfered with by unequal demands upon its reserve force—as it is apt to occur, *e.g.,* in overexertion, intercurrent diseases and the like—we can readily formulate a plan of treatment which will, at least for a time, amply meet with the aforementioned indication. Parents should be given to understand that the treatment of compensating heart disease is principally prophylactic and hygienic, and that its success is commensurate with the degree of co-operation on the

Maintenance of compensatory hypertrophy.

part of the patient as well as those guiding his destiny when the heart is at its best.

Care during convalescence.

Convalescence from acute or recurrent heart disease calls for very careful attention. Too early attempts at walking or standing are apt to prove disastrous, not rarely leading to sudden dilatation of the heart, perhaps, with fatal issue. Beginning with gradual raising of the head and shoulders, and watching its effect upon the patient's heart-action—strength and rhythm—we may gradually allow greater liberties, provided slight exertion is unattended by detrimental influences. In severe cases of valvular heart disease it is usually not safe to permit the patient to be out and around in less than three months. A sojourn in a quiet inland resort is very helpful to recovery.

Ample nutrition.

Non-alcoholic tonics.

A heart with crippled valves demands an adequate supply of healthy blood in the coronary arteries. This is best secured by suitable nutrition and a rational mode of living. The diet must be appropriate to the age of the patient, at all ages milk forming the principal food-ingredient. A vegetable diet with small quantities of light meats is suitable in most cases. Liquors and stimulants of all kinds should be avoided, administering instead nutrient tonics such as malt and cod-liver oil, with or without small quantities of iron and arsenic, etc.

Outdoor air.

Avoidance of physical and mental overexertion.

Especial attention should be paid to the action of the bowels, kidneys, and skin. Daily cool sponging followed by gentle massage is very invigorating. Warm clothing is essential, but unnecessary coddling of the patient should be interdicted. Weather permitting, the child should be kept outdoors from nine in the morning until about five o'clock (later in the summer) in the evening, allowing him to participate in all such amusements as will not call for undue exertion. Racing, jumping, football, and baseball playing and swimming should be forbidden. Light athletic exercise is useful if it gives rise to no undue fatigue, or disturbance of compensation. Passive exercise, in the form of massage, is highly to be recommended. The question of how much brainwork a patient with poorly compensating heart disease is to be permitted to do cannot be decided offhand to apply to all cases. Its effect upon the general health of the patient must be watched, and changes in the curriculum promptly made if headache, insomnia, anemia, debility, excessive nervous irritability, and the like make their appearance.

It is of very vital importance to obviate intercurrent diseases, Prevention of communicable diseases.

especially infectious diseases, such as scarlatina, articular rheumatism, etc., which are apt to reinfect the endocardium, and aggravate the patient's condition. If such diseases prevail it is imperative, whenever practicable, to isolate the child, or remove him to a place where he will be least exposed to infection. For fear of contracting contagious diseases, patients in good financial circumstances should be kept from visiting public or private schools and preferably be instructed at home.

Particular attention should be paid to incipient symptoms of tonsillitis, "growing pains," etc.—forerunners of rheumatism. In these conditions the salicylates should be resorted to early to prevent graver rheumatic manifestations.

With every appearance of indisposition the patient should be put to bed, and kept there until every vestige of the malady has abated.

In intercurrent febrile diseases the heart demands very careful watching, and in the presence of any disturbance immediate treatment.

STAGE OF FAILING COMPENSATION.

Varying with the inherent strength of the patient, the severity of the lesion and the precautionary measures employed, compensation may be maintained for a shorter or longer time—weeks, months or years. However, it is only a question of time when compensation ruptures. As previously mentioned, the breakdown may be only temporary—readily yielding to a few weeks of rest, careful feeding, possibly requiring also a few doses of digitalis—and recur on several occasions. But sooner or later the heart muscle gives way—the pulse becomes feeble and irregular, the breathing deep and difficult, the urine diminished in quantity and the general condition of the patient greatly impaired. Here rest in bed is indispensable, but this alone is not sufficient to restore compensation. We have to resort to cardiac stimulants to strengthen the heart muscle and to regulate its beat, and to vasodilators, with each ventricular contraction, to allow the blood to flow in the arteries without resistance. Various drugs are being recommended for this purpose, but none meet the indications with the same degree of certainty as digitalis, and the iodids. In incipient failure of compensation we usually begin, for every two years of the child's age, with half a grain of the sodium iodid and a quarter of a dram of the infusion of digitalis, to be

Signs of heart-failure.

Digitalis and the iodids.

Strophanthus, caffeine.

repeated every six hours, and as the disease advances increase the doses proportionately up to one grain of the iodid and one dram of the digitalis. The cumulative action of the digitalis should be borne in mind, and its administration discontinued if untoward symptoms arise. In this event, or where digitalis is not well tolerated by the stomach, we may substitute strophanthus, caffeine sodium benzoate or spartein sulphate instead. The latter two remedies have the advantage that they may be safely given hypodermatically if irritability of the stomach precludes their administration by mouth.

Symptomatic treatment.

In the early attacks of failure of compensation the effects obtained from the simple mode of treatment just outlined are often entirely beyond expectation. Sometimes within but a very few days the urine greatly increases in quantity, the edema disappears, the dyspnea ceases, the distressing cough abates—in short restoration of compensation is apparently complete. In the later stages of compensatory failure, however, the treatment by means of rest, good food, the iodids and digitalis fails to assert its magic influence. We have to resort to symptomatic medication, especially with the view of relieving suffering. In this respect the treatment is the same as that employed in adults, morphine with atropine being the most potent remedy at our command.

℞ Strychninæ sulph.	gr. ⅛	0.008
Natrii iodidi	gr. xvj	1
Inf. digitalis fol.	℥j	30
Syr. altheæ q. s. ad	℥ij	60

M. Sig.: ʒj t. i. d. for a child 4 years old. (Alterative heart-tonic.)

℞ Syr. ferri iodidi	ʒiij	12
Syr. aurantii q. s. ad	℥ij	60

M. Sig.: ʒj every four hours for a child 4 years old. (Between "heart attacks.")

℞ Liq. ferri et ammonii acetatis,		
Inf. digitalis fol. āā	℥j	30

M. Sig.: ʒj every four hours for a child 4 years old. (When dropsy is present.)

℞ Tr. digitalis,		
Tr. strophanthi āā	ʒij	8

M. Sig.: Gtt. v every four hours for a child 4 years old. (In marked heart-dilatation with irregularity.)

℞ Strychninæ sulph.	gr. ⅕	0.003
Caffeinæ natrii benzoatis	gr. xij	0.8
Aq. destil.	ʒij	8

M. Sig.: Gtt. x, hypodermatically, p. r. n. for a child 4 years old. (Quick stimulant.)

CHAPTER XI.

Diseases of the Kidneys, Bladder, etc.

NEPHRITIS ACUTA.

Common in communicable diseases.

ACUTE nephritis is most frequently met in association with acute infectious and contagious diseases, especially scarlatina, diphtheria, and pneumonia. Less frequently it occurs as a result of exposure to wet and cold; of structural alterations of the skin, *e.g.*, extensive burns; of ingestion of certain irritants to the kidneys, *e.g.*, cantharides, potassium chlorate, aspidium, etc., and, finally, not rarely it is observed in infants suffering from gastroenteric affections. The aforementioned causes usually operate upon both kidneys, so that both kidneys are equally affected. The lesion may, however, remain limited to one kidney where the disease is caused by direct, unilateral trauma. The seat of the kidney lesions varies somewhat with the cause. For example, the glomeruli (*glomerular nephritis*) are most severely involved in scarlatina, while in diphtheria we most commonly find degenerative changes in the renal tubules (*degenerative or parenchymatous nephritis*). But no particular form of acute nephritis is peculiar to a given cause. In severe cases the kidneys are greatly increased in volume and weight. The surface is smooth and the capsule readily removable. The renal cortex is either uniformly reddened or pale and mottled with red. The tubuli uriniferi are partly or completely obstructed by large granular epithelial cells, blood-corpuscles and fibrin. In the early stage of the disease the interstitial tissue shows no alteration; in protracted cases, however, this tissue may suffer very severely. In this event the process is often spoken of as productive or *interstitial nephritis*.

Parenchymatous.

Interstitial.

Consonant with the etiological factors we distinguish a primary and secondary form of acute nephritis, but, except for some slight difference in the onset (it being more sudden in primary nephritis), the symptomatology is practically the same in both varieties. The child complains of backache, headache, nausea and chilliness, occasionally vomits, and, in severe forms, shows

Edema.

Albuminuria and casts.

other symptoms of grave constitutional disturbance. Not infrequently attention to the illness is not attracted until the appearance of puffiness of the eyelids, or, especially in infants, the occurrence of partial or total suppression of urine with or without uremic symptoms. Examination of the urine discloses more or less marked alteration in its constituents. Chemically the urine almost invariably reveals the presence of a variable amount of albumin, and, microscopically, casts of all sorts, especially hyaline, red and white corpuscles, epithelium, detritus, etc. The urine is usually acid, and its specific gravity high, the latter being, of course, most marked when the quantity is very small. The secretion of urea is diminished. In severe inflammation of the kidneys the urine contains a large quantity of blood (hemorrhagic nephritis), and is dark-red or smoky in color.

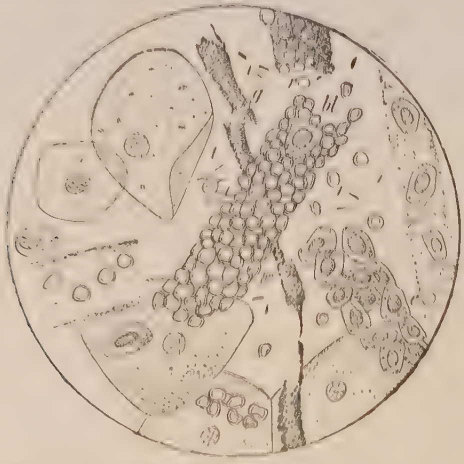

Fig. 139.—Acute Hemorrhagic Nephritis. × 350. Small and large squamous epithelium, hyaline casts (at the margin). *g*, Finely granular cast. *bl*, Red blood-corpuscle cast. *e*, Tubular epithelium (arranged in cast form). Here and there are blood-corpuscle rings ("shadows" [ghosts]). (*Lenhartz* and *Brooks*.)

Hematuria.

As already alluded to, the onset of nephritis often escapes detection. This is especially true in the secondary form. Hence the importance of systematic examination of the urine during the course of acute communicable diseases. It is well to remember, however, that not every albuminuria is of nephritic origin. A small quantity of albumin and a few casts are not rarely found in acute febrile diseases (*e.g.*, in the beginning of scarlatina) without kidney lesions and are only transitory in nature.

Cases running a favorable course begin markedly to improve after about two weeks. The albumin diminishes; the urine increases in quantity, becomes light and clear, and the microscopic abnormal constituents subside. Edema, if present, is slight, and usually limited to the eyelids and rapidly disappears with the improvement of the other symptoms.

Benign cases.

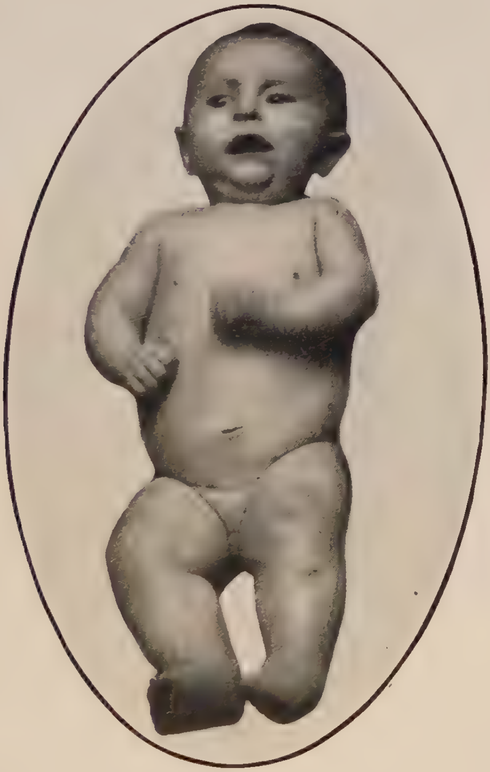

Fig. 140.—Acute Nephritis with General Anasarca in a Child 4 Months Old. (*Sheffield.*)

Less favorable cases are of longer duration. From day to day the edema assumes wider dimensions, involving the dorsi of the feet, the legs, the genitalia, and, if not checked, the serous effusion may rapidly fill the abdominal and thoracic cavities. In the majority of instances, however, gradual recovery from the immediate attack occurs, although in these cases a relapse must always be apprehended.

Malignant cases.

Oliguria up to anuria. Uremia.

Another group of cases is characterized by great diminution of urine (oliguria) or total suppression and consecutive uremia. The latter is manifested by intense headache, dizziness, vomiting, dimness of vision up to total blindness, disturbance of hearing, slight twitching up to repeated attacks of severe convulsions, slow, irregular pulse, dyspnea, somnolence, sopor and possibly coma and death.

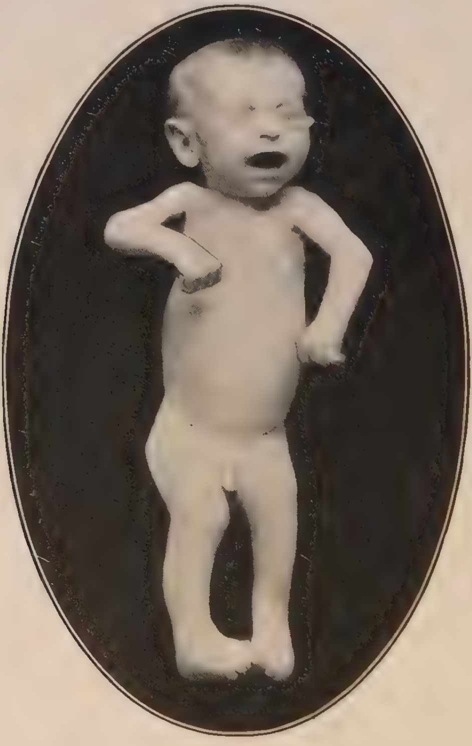

Fig. 141.—Same case as Fig. 140. Three weeks later. (*Sheffield.*)

Scarlatinal.

The incipient symptoms of nephritis offer no reliable indications as to the further course of the disease. Scarlatinal nephritis, for example, ushers in with vomiting, intense headache, convulsions, local or general dropsy, and yet clears up completely within two or three weeks; and, conversely, nephritis may set in insidiously, apparently entirely free from any alarming symptoms, and, nevertheless, proceed a very protracted course and possibly lead to permanent degeneration of the kidney structures.

Relapse.

Furthermore, relapses may complicate matters often when recovery is imminent.

The prognosis, therefore, should always be guarded, even though the general condition of the patient is good. Even in mild cases untoward complications are apt to supervene. Serous effusions in internal cavities are not rare. This is true especially of ascites, less frequently of pleural or pericardial effusions. The heart rarely escapes involvement. Hypertrophy of the heart is quite common, and if the nephritis runs a protracted course dilatation of the heart may prove a very dangerous complication, particularly in view of the secondary pulmonary edema, which is very prone to occur in such cases, and often prove fatal. Extensive anasarca with scanty urine, especially if ascites is associated with hydrothorax, greatly mars the prognosis. As further complications we may mention uremia, pneumonia, edema of the glottis, severe intestinal catarrh, more rarely peritonitis, pericarditis and endocarditis (more frequent in scarlatinal nephritis). Notwithstanding, however, the great array of complications, immediate death from acute nephritis, especially from the primary variety, is not common. The death rate ranges between five and twenty per cent.—the variation depending upon the primary cause, mode of treatment and severity of the complications. A great many patients who survive the acute stage remain invalided for life. As we shall see later, gradual transition from acute into chronic nephritis is not of uncommon occurrence. Convalescence is often prolonged for weeks and months, and even without permanent injury to the kidneys albumin may recur in the urine from time to time for a period of a year or two or longer and continue to undermine the child's constitution.

Ascites.

Cardiac hypertrophy.

Pulmonary edema.

Complications.

Chronic course.

Every case of nephritis, be it ever so mild, should be taken seriously, and kept under strict observation not only during the active stage of the affection but for many months after. During the acute stage perfect rest in bed should be enjoined and the diet limited to bland articles of food free from salt, preferably milk in moderate quantity with strained oatmeal or barley, zwieback with sweet butter, stale bread with a little apple sauce, and occasionally a little chicken soup. The partaking of water should be restricted to a few tumblerfuls of Vichy or lithia water per day. As the condition improves the dietary may be augmented by the addition of freshly boiled—without salt—vegetables, such as carrots, spinach, cauliflower, fresh green peas, etc., stewed fruit, and freshly boiled whitefish. The bowels should be kept open by an occasional dose of calomel followed by citrate of magnesia

Rest.

Restricted diet.

Laxatives.

and by daily high intestinal irrigation. Where the excretion of urine is greatly reduced and the dropsy marked, energetic measures to relieve the kidney should be instituted without delay. This should be attempted, not, as is frequently advised, by means of active diuretics—which only help to increase the renal congestion—but by stimulating the activity of the skin and allaying the irritation of the kidney. For this purpose we resort to hot packs (105° F.), hot baths (103° F.), and hot colon flushing (110° F.). These may be repeated every six hours. Perspiration may be stimulated by small quantities of hot water, or hot lemonade. In hemorrhagic nephritis small doses of ergot act beneficially. Camphor will be found valuable to counteract collapse, and should be administered hypodermatically in the form of sterilized camphorated oil. Excessive irritability of the nerve system should be combated by means of the bromids and chloral.

Hot baths; colon flushing.

Camphor.

By carefully following the aforementioned directions, uremia is of rare occurrence. Uremic convulsions should be controlled by chloroform inhalation, hypodermatic injection of morphine and atropine (for a child two years old gr. $\frac{1}{32}$ of morphine, and gr. $\frac{1}{500}$ of atropine, if necessary to be repeated once after two hours), and, where these therapeutic measures fail, by lumbar puncture.

Arrest of uremia.

Children recovering from nephritis should not be exposed to the ill effects of overfeeding, overexertion, and exposure to marked atmospheric changes. They should wear light woolen underwear, and, financial means permitting, should spend the winter following an acute attack of nephritis in a warm climate.

After-treatment.

To overcome the remaining anemia, iron and cod-liver oil will be found of service.

NEPHRITIS CHRONICA.

In the majority of instances chronic nephritis develops as a sequel of the acute affection of the kidneys. The parenchyma or interstitial tissue or both remain permanently impaired. On the one hand, we may find the kidneys greatly enlarged, the cortical portion increased in volume, its surface white or pale-yellow—large white kidney or parenchymatous nephritis. On the other hand, the whole organ is reduced in size, the capsule firmly adherent, and the surface irregular and nodular—the granular or cirrhotic kidney, or interstitial nephritis. Amyloid

Large, white kidney.

Cirrhotic kidney.

degeneration is another form of chronic nephritis in childhood. It is usually associated with amyloid degeneration of the liver and spleen, and ordinarily occurs secondarily to suppurative processes of the bones or joints. Occasionally chronic nephritis is encountered in connection with congenital malformations of the kidneys, or as result of hereditary syphilis, tuberculosis, and heart disease.

Findings in urine.

In the early stages of chronic nephritis the diagnosis rests principally upon the chemic and microscopic findings in the urine. In parenchymatous nephritis the quantity of urine is normal or diminished, the specific gravity normal or increased, the albumin content high, and the color cloudy, brownish yellow or bloody. In interstitial nephritis the quantity of urine is increased, the specific gravity low, the albumin content low (occasionally no albumin), and the color clear, and pale. In amyloid degeneration the urine is rich in serum-albumin and globulin. Its quantity is often increased. Casts in the urine are present in all varieties.

Anemia.

Heart symptoms.

With further advance of the disease, the appearance of profound anemia, digestive and respiratory disturbances, local and general dropsy, and cardiac debility readily discloses the underlying condition. Toward the end of life the symptoms resemble greatly those of non-compensating heart disease.

Parenchymatous nephritis offers the worst prognosis, death usually setting in within a year from the appearance of the secondary symptoms. The course of interstitial and amyloid nephritis is much more protracted and cases of amyloid kidney are on record which markedly improved on removal of the suppurative bone affection, but complete recovery is practically out of question.

Salt-free diet.

Foods to be avoided.

Under suitable treatment (except in the parenchymatous variety) life may be prolonged for many years. As in acute nephritis, the diet should be free from salt, but otherwise more liberal. Older children may live on a mixed diet; the following food-stuffs, however, are to be exempt from the list: Liver, ham, brains, kidneys, beef-juice and beef-extract, coffee, liquors and spices. All meats, eggs and fish should be taken sparingly. Whenever possible, the child should live in a warm climate. Outdoor life and very *light* exercise are desirable. Daily warm baths with gentle massage act beneficially. With appearance of dropsy, dyspnea, or other grave symptoms, the patient should be put to

Rest. bed and treated in the manner outlined under "acute nephritis" (*q. v.*).

Hematinics. Hematinics, in small doses, and other tonics in the form of cod-liver oil, nux vomica and digestants are in order as necessity arises. Excessive dropsical effusions should be relieved by active catharsis, alkaline diuretics, and heart stimulants, in addition to the therapeutic measures recommended in dropsy accompanying acute nephritis.

NEPHROLITHIASIS

(Stones in the Kidney; Renal Calculi).

Sudden attacks. Renal calculi in children give rise to symptoms identical with those observed in adults. Thus, sudden attacks of pain in the lumbar region, radiating downward along the course of the ureters, groins, and, in the male, to the testicles. The attacks are usually associated with nausea, vomiting and convulsions and often collapse. The urine is passed frequently, in small quantities, and contains blood- and pus-cells. Hematuria; pyuria. The urine, however, may appear normal if it is excreted from the healthy kidney only, or there may be complete anuria if both ureters are simultaneously obstructed. Where the stones remain impacted in the ureter, the condition is apt to become very grave in consequence of supervening hydronephrosis, pyonephrosis or pyelonephritis. Hydronephrosis. In this event we are often obliged to resort to surgical interference. Otherwise symptomatic treatment usually suffices to effect marked improvement or even a cure. Alkalies should be administered in uric acid concrements, sodium phosphate in oxalic acid, and citric acid and acetic acid in phosphatic concrements. Specific medication. The diet should be bland (avoidance of meat), and metabolism enhanced by digestives, mild laxatives, moderate exercise, hydrotherapy and massage. Anodynes. To relieve an attack we resort to anodynes (morphine and atropine hypodermatically), hot baths and hot poultices.

An X-ray examination is often decisive in the diagnosis between nephrolithiasis and appendicitis, with which affection the former may be confounded.

PYELITIS, PYELONEPHRITIS, PYELONEPHROSIS.

Inflammation of the pelvis of the kidney and contiguous structures with consecutive suppuration may occur as a result of Renal stones. direct injury of the lining mucous membrane, *e.g.*, renal stones;

as a sequel of infectious diseases, such as scarlatina, diphtheria, variola, or pyemia, or by extension of a suppurative process from the neighboring tissues or organs, *e.g.*, perinephritic abscess, cystitis, colicystitis, purulent vulvovaginitis. It is also met in connection with congenital malformations of the kidneys or ureters, renal tuberculosis and tumors. The pyelitis may be unilateral (when due to a local cause) or bilateral.

Colicystitis.

The symptomatology of pyelitis varies greatly with the cause and the course it pursues. In acute cases there are rigors, high and fluctuating temperature, frequent and scanty urination, pain in the lumbar region, and, above all, pyuria. The morphological constituents of the urine vary with the degree of involvement of the kidneys, ureters and bladder. Cases proceeding a chronic course are ordinarily free from febrile excursions. Pyonephrosis often gives rise to a palpable tumor. There are a number of other symptoms which vary with the primary or secondary disease.

Rigors, fever, pyuria.

Tumefaction.

Where the cause is removable, and prompt treatment is instituted, the pyelitis may entirely disappear and leave the kidney uninjured. Otherwise the prognosis, as to complete recovery, is bad. The prognosis as to life depends entirely upon the exciting cause and complications, nephritis and exhaustion forming the principal sources of danger.

The aim of treatment, therefore, should be to avoid the latter by early elimination of the fundamental disease, and prevention of recurrence of attacks. The details of such treatment are fully outlined when speaking of the diseases in question. Otherwise the treatment is symptomatic. The urine should be rendered alkaline, and as aseptic as possible. This is best accomplished by a liberal supply of water, alkaline diuretics and hexamethylenamin. Pyonephrosis calls for surgical interference.

Prevention of recurrence.

Operation.

HEMOGLOBINURIA.

Hemoglobin or methemoglobin in the urine is occasionally observed in infants and older children, either as a result of poisoning by phosphorus, potassium chlorate, carbolic acid, etc., or in connection with severe burns, acute and chronic infectious diseases, such as exanthemata, malaria, and hereditary syphilis. The urine is mahogany-brown or black in color, greatly resembling bloody urine. Microscopically, however, it shows the presence

Toxic drugs.

Smoky urine.

No blood. of blood coloring substance only, but no blood-corpuscles. The spectroscope discloses bands of hemoglobin. The attacks of the hemoglobinuria are of brief duration (sometimes last but a few hours), and are manifested by debility, chilliness, cyanosis, and sometimes high fever. These symptoms disappear as the urine clears up. Occasionally the hemoglobinuria appears in paroxysms (*paroxysmal hemoglobinuria*) without any discernible cause or after exposure to cold or undue fatigue.

Paroxysmal.

By rest in bed, liberal supply of liquids, and attention to the exciting causes, the hemoglobinuria subsides without any serious consequences. (See also "Epidemic Hemoglobinuria," page 182.)

ORTHOTIC, CYCLIC OR FUNCTIONAL ALBUMINURIA.

Albumin absent when patient is at rest.

As the term (orthotic = standing up) indicates, the disease is characterized by the presence of albuminuria after the patient has been up and around, and by its absence while he is perfectly at rest. It is observed especially in delicate children of from five to fifteen years old, and seems to have nothing in common with organic kidney disease. A family predisposition has been traced in some cases, and a history of scarlatina and diphtheria in others. The urine is free from abnormal morphological constituents—the opposite being the case in true renal disease.

Under suitable treatment, which is essentially the same as in the early stage of chronic nephritis, the albuminuria often disappears for a time, but may return after a shorter or longer interval (intermittent form). Notwithstanding the continuance of the albuminuria for many years, the system is very little affected by it, and the prognosis as to life is good. Transition of cyclic albuminuria into nephritis, however, is on record.

TUMORS OF THE KIDNEY.

Benign.

Aside from tuberculosis and syphilis, which have been discussed elsewhere, the kidneys are occasionally the seat also of benign and malignant neoplasms. The *benign* tumors (adenoma, fibroma, lipoma, cysts, etc.), owing to their very slow growth, generally escape observation, and are often found post mortem in children who, during life, never manifested signs of kidney growths. To a great extent this is true also of *malignant* tumors (sarcoma, carcinoma, myosarcoma and adenosarcoma) in their

Malignant.

early stages of development, since the tumor is barely palpable and the two additional characteristic symptoms of malignant kidney growths (*i.e.*, hematuria and cachexia) are present in only a small percentage of such cases and is encountered also in a number of other wasting diseases. **Hematuria.**

In the beginning the tumor may be felt only in the loin; within a few months, however, it is found to have spread in all directions, displacing the liver, spleen, lungs and intestines, and to occupy the entire abdominal cavity (see Fig. 142). Not rarely it forms secondary metastases in the other kidney, in the liver, spleen, intestines and retroperitoneal glands, and, by pressure upon the ureter, may give rise to hydronephrosis. **Palpable tumor.** **Metastases.**

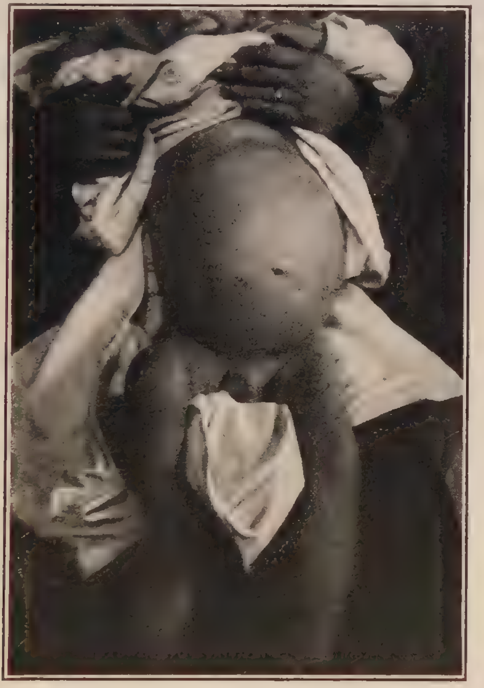

Fig. 142.—Sarcoma of the Kidney (child 27 months). The tumor occupied almost the entire abdomen. (*Sheffield.*)

Unless operated upon early—which treatment should invariably be recommended—the children usually succumb to progressive emaciation and exhaustion within about a year from the time the tumor makes itself felt. As the majority of growth is of antenatal origin nothing can be done in the way of prophylaxis. **Early operation.**

CYSTITIS.

Primary. Inflammation of the bladder may occur as a primary or secondary disease. Primary cystitis is extremely rare in children, more especially in infants, since the principal cause—direct mechanical injury of the mucous membrane by surgical instruments or other foreign bodies—is but very rarely operative in young children. On the other hand, secondary cystitis is of comparatively frequent occurrence, and may arise from a great many causes, the most important of which being infectious diseases (diphtheria, scarlatina, etc.), kidney and bladder diseases (calculi, pyelitis, tuberculosis, tumors, etc.), cerebrospinal affections (atony and overdistention of the bladder with consecutive inflammation by decomposed urine), intestinal diseases (invasion of the bladder by colon bacillus—*colicystitis*), and diseases of the vagina and urethra, especially of gonorrheal origin (by extension of the inflammation). Cystitis may follow chemical irritation (overdoses of cantharides, balsams, liquors, etc.), exposure to cold (sitting on cold stones, etc.) and direct external violence.

Secondary.

Colicystitis.

The lesions in the bladder may range from simple localized redness to extensive ulceration of the mucous membrane and pseudomembranous deposit. In cases of long standing the inflammation is prone to spread to the ureters and kidneys. In chronic cystitis the mucosa assumes a gray, pigmented color, becomes greatly hypertrophied, and covered by mucopurulent masses.

Extension upward.

In accord with the severity and extent of the lesion cystitis may be manifested by mild or grave symptoms. The latter are most pronounced in primary cases, in those associated with infectious diseases (*e.g.*, diphtheria), and in infection by the colon bacillus. In mild cases the symptomatology consists of painful and frequent micturition, sensitiveness over the region of the bladder, sometimes rectal tenesmus and excoriation of the urethral orifice and of the contiguous structures. The urine is voided in small quantities, sometimes only a few drops at a time, and contains mucous shreds, bladder epithelium, pus-corpuscles, blood-corpuscles, and numerous bacteria. The urine is neutral or alkaline, cloudy and dark red, and may contain pieces of membrane if the cystitis is of diphtheritic origin. In colicystitis the urine is usually acid in reaction, and in addition to the aforementioned constituents presents traces of albumin. The constitutional symptoms are slight. Severe forms of cystitis, especially colicystitis, give rise to marked constitutional disturb-

Strangury.

Blood.

Acid urine in colicystitis.

Constitutional symptoms especially in colicystitis.

ances, such as vomiting, chills, irregular fever, and sometimes convulsions (particularly if anuria exists). The local symptoms also are much more pronounced. If left to run its course, the condition is not rarely aggravated by the concurrence of nephritis, which may lead to a fatal termination.

As it is not always possible in the beginning to foresee the eventual course of the disease, and as the tendency even of mild cases towards chronicity is great, it is essential not to trifle with the affection, but promptly to employ all such therapeutic measures as will insure its early arrest and ultimate cure. The patient should be put to bed and on a mild diet (milk and Vichy water, milk-gruel, chicken soup, eggs, cereals and bread). All spices, alcoholic beverages, coffee and tea should be prohibited. To relieve pain, hyoscyamus is the remedy *par excellence*. It may be combined with acetate of potash and small doses of hexamethylenamin. Warm Priessnitz compresses are also of value. Where the pain persists, a suppository of codeine and extract of belladonna will be found to act well. With subsidence of the acute symptoms—usually after a week or two—it is advisable to begin to irrigate the bladder (under the most careful aseptic precautions) with a warm solution ($\frac{1}{2000}$ or $\frac{1}{1000}$) of nitrate of silver or potassium permanganate. From half a pint to one quart of the solution may be used for each treatment, and the irrigation may be repeated once a day or every other day.

Restricted diet.

Hyoscyamus.

Codeine.

Irrigations.

Under this method of treatment the majority of cases of cystitis will recover in from four to eight weeks—provided, of course, the primary cause can be detected and removed.

Transition of acute cystitis into chronic is by far less common in children than in adults. The possibility of the disease being tubercular should always be borne in mind (see page 370).

Tendency to chronicity.

℞ Kalii acetatis	ʒj	4
Ext. hyosciami fl.	gtt. xvj	1
Inf. uvæ ursi	℥j	30
Aq. anisi	q. s. ad ℥ij	60

M. Sig.: ʒj, in water, every three to six hours for a child 3 years old.

℞ Hexamethylenaminæ	gr. xvj	1
Natrii benzoatis	ʒss	2
Ext. belladonnæ fl.	gtt. iv	0.3
Mucilaginis ulmi	ʒiv	15
Aq. fœniculi	q. s. ad f℥ij	60

M. Sig.: ʒj, in water, every four hours for a child 3 years old. (In colicystitis.)

See also "Biologic Therapeutics," page 96.

VESICAL CALCULI
(Stones in the Bladder).

Strangury. **Interrupted flow of urine.** **Impacted stone.**

Bladder stones sooner or later give rise to the following characteristic symptom-complex: Vesical and often rectal tenesmus, strangury, partial or complete retention or incontinence of urine, difference in the force of the stream of the urine with change in posture of the patient, and, after a protracted course, the usual symptoms of cystitis (*q. v.*). The urine may reveal the presence of either phosphate stones (phosphate and carbonate of lime, magnesia), oxalate stones (oxalate of lime) or urate stones (uric acid). Small concrements may escape with the urine; large ones, however, are apt to become impacted in the urinary canal and cause intense pain and grave nervous symptoms, *e.g.*, convulsions.

X-ray.

The diagnosis is based upon the aforementioned manifestations, upon feeling the stone in the bladder by rectal digital examination or by a sound introduced into the bladder, and upon an X-ray examination.

Anodynes.

The development of stones may frequently be prevented by a bland diet (no meats), ample supply of water, and attention to the bowels. In cases of long standing operative interference is indispensable. Painful symptoms are relieved by means of hyoscyamus.

SPASMUS VESICÆ, DYSURIA, ISCHURIA (ANURIA).

Calculi. **Uric acid infarct.**

These conditions are etiologically correlated. In the majority of instances they are the result of vesical calculi, blood-clots obstructing the urinary flow, phimosis, paraphimosis, vulvitis and vaginitis, cystitis, uric acid infarcts (in the newly born), sudden chilling of and injury to the lower portion of the abdomen, nerve affections (functional or organic), and priapism (in the male).

The treatment varies with the original cause. An attack is usually relieved by a hot bath, a suppository of codeine and extract of belladonna, and the administration of diuretics, such as sweet spirits of niter and triticum repens.

℞ Kalii citratis	ʒj	4
Ext. hyosciami fl.,		
Ext. triticum repens fl.	āā ʒss	2
Syr. simplicis	ʒiv	15
Aq. destil.	q. s. ad f℥ij	60

M. Sig.: ʒj, in water, every three to six hours for a child 3 years old.

ENURESIS.

It is customary to distinguish two varieties of enuresis in children: Enuresis diurna and enuresis nocturna. The first variety is but rarely met in children, capable of differentiating right from wrong, excepting in those who willfully "wet" themselves, or in congenital deficiencies. The second variety, on the other hand, occurs in a very great number of children, regardless of age, sex, intelligence or social conditions. The child may wet itself one or more times every night, or at intervals of days or weeks; in the last event, it is usually due to willfulness, excessive drinking, or faulty diet. An inherited tendency and neurotic disposition seem to play an important part in the causation of enuresis, although the latter may exist independently of either of these causes in children apparently perfectly healthy.

Diurna and nocturna.

The causes of enuresis may conveniently be arranged in two classes:—

1. FUNCTIONAL.

The cases due to functional causes are purely neurotic in character. The urine is voided involuntarily either owing to *atony* of the sphincter vesicæ, or to a *spasmodic* condition of the detrusors vesicæ. In both cases there is a functional disturbance in the nervous apparatus of the urinary system. It is usually found that enuresis due to *atony* is associated with general debility, and often follows a protracted course of an exhausting disease. On the other hand, enuresis due to "spasm" is usually found in children who are irritable, who present an increased patellar reflex, are easily frightened, are subject to pavor nocturnus and similar nervous conditions.

Atony.

Spasm.

2. ORGANIC.

The greater number of cases arise from organic troubles. The child may suffer from:—

Organic disease of the spinal cord; cystitis; phimosis or paraphimosis (in the male); hypertrophy of the clitoris or adhesion of the prepuce (in the female); masturbation; undescended testicle; hernia; worms; vesical and renal calculi; tumors in the bladder; excessive quantity of lithiates or phosphates; constipation and accumulation of feces; epi- or hypo-spadia; fissure ani; vulvovaginitis; diabetes, gonorrhea, simple or gonorrheal proctitis.

Local and systemic affections.

Regular habit.

(In the treatment of enuresis it is of greatest moment to systematically examine the patients for the organic diseases just enumerated and to endeavor to eliminate every symptom suspicious of organic disease.) In absence of organic causes there is evidently a neurotic case to be dealt with and the treatment must be adopted accordingly. Patient if old enough should be instructed not to abstain from micturition when called upon by nature to do so, and small children should be trained to void urine about every three hours, and not be permitted to withhold the urine for a longer period. ✓This is very important, for it is often overdistention of and decomposition of the urine in the bladder that prove the primary cause of the subsequent secondary etiological factors (atony or hyperesthesia of the bladder, presence of concretions, cystitis, etc). ✓It is also advisable to encourage drinking of water in cases of enuresis due to concretions, cystitis, or gonorrhea, but to forbid it in other cases. ✓The patient is not to be permitted to sleep on his back, and it is often of advantage to raise the foot of the bed in such a manner that the child's trunk and head lie deeper than the pelvis.

Tonics.

In enuresis due to *atony* a general constructive treatment is indicated. Plenty of good nourishment, change of air, cold spinal douches, medicinal tonics and electricity are usually effective in bringing about a cure. A moderate galvanic current is usually best; one pole is applied to the symphysis or rectum, the other to the perineum. The following mixture is often very serviceable:

℞ Ext. ergotæ	fʒiij	12
Ext. rhus tox.	fʒj	4

M. Sig.: Five to 10 drops every four to six hours to a child 6 years old.

Antispasmodics.

In incontinence of urine associated with *hyperesthesia* of the collum vesicæ or spasm of the detrusors, an antispasmodic treatment is indicated, consisting of hot sitz-baths, avoidance of irritating food or drinks and the administration of either ext. belladonna or hyoscyamus. I usually prescribe the following:—

℞ Ext. hyoscyami	ʒss	2
Natrii bromidi	ʒj	4
Aquæ anisi	℥j	30
Syrupi simplicis	q. s. ad ℥ij	60

M. Sig.: One teaspoonful every four to six hours to a child 6 years old.

Counterirritation by means of sinapisms over the lumbosacral regions often does well, and if everything fails this class of cases is occasionally cured by gradual dilatation of the posterior urethral canal.

As to the treatment of enuresis from organic causes, nothing more will be said here than that each case must be treated as an individual disease in accordance with its etiology.

Moral suasion.

Remonstrance, severity and moral suasion will often cure cases of enuresis of nervous origin or those which continue from mere habit long after removal of the original cause.

VULVOVAGINITIS.

Notwithstanding recent advances in bacteriology and microscopy, the profession is not as yet in accord as to the exact nature of vulvovaginitis in children. Some physicians still doubt the fact that most cases are due to the gonococcus of Neisser and are highly contagious, but tenaciously cling to the "scrofulous" theory of the disease and recommend tonics to combat it. As a result, innumerable cases run at random, leaving sources of contagion in public schools and baths, homes, and hospitals, with apparently no one in authority to check the further spread of the affection.

Clinically vulvovaginitis may be classified as follows:—

1. Catarrhal vulvovaginitis, which is generally due to (*a*) lack of cleanliness or (*b*) chemical irritation.

2. Traumatic vulvovaginitis, which is caused by (*a*) masturbation (?), (*b*) mechanical injury, or (*c*) indecent violence.

3. Parasitic vulvovaginitis, which is due to (*a*) oxyurides, (*b*) saprophytes, or (*c*) pathogenic bacteria, especially the gonococcus.

Catarrhal.

The first variety of vulvovaginitis is usually met in poorly nourished children of overcrowded tenement districts, who receive a thorough cleansing on very special occasions only. As a rule, these cases begin with vulvitis, the vagina becoming gradually involved by extension of the inflammation. Catarrhal vulvovaginitis is not always limited to the very poor, and the physician need not hesitate to suspect dirt even under the most elaborate apparel.

Dirt.

Chemical irritation.

This variety of vulvovaginitis is also frequently observed in children whose genitalia are exposed to excessive wetting by irritating, decomposing secretions, and excretions—sweat, diarrheal stools, hyperacid urine—and to undue pressure and friction.

In former years, when bicycle riding was a national fad, vulvovaginitis was not rarely met in assiduous bicycle riders, undoubtedly as a result of the aforesaid causes.

Traumatic. The consideration of the second, traumatic, variety of vulvovaginitis does not, strictly speaking, belong to the domain of medicine, except as regards the treatment. We are dealing here with faulty habits and criminal traits which deserve serious attention on the part of teachers, the clergy, and jurists. However, as it is the physician who is usually consulted first, a few points of information will prove useful to him, particularly as a warning not to be too hasty in expressing a positive opinion.

Masturbation. I believe that entirely too much stress is being laid by some authors upon masturbation as an etiological factor of vulvovaginitis. It is much more probable that masturbation is a result rather than a cause of it, the undoubtedly existing irritated state of the erectile tissue inducing that bad habit.

Foreign bodies. The presence of foreign bodies in the vagina is not infrequently found to be the cause of vulvovaginitis. While some girls will introduce foreign bodies in the vagina with lascivious intent, the great majority of foreign bodies, *e.g.,* safety pins, will find their way in the vaginal canal accidentally, and should always be looked for, particularly in cases of long standing.

Rape. Occasionally cases of vulvovaginitis are encountered which are the result of indecent violence. The purulent discharge is either non-gonorrheal or gonorrheal, the latter only if the criminal who attempted rape had at the time been suffering from gonorrhea. It is well to remember that not every case of vulvovaginitis reported to be due to rape is really such, and unless the vaginitis is associated with actual penetration of the hymen and concomitant signs of inflammation due to violence, the physician should be very cautious in venturing a positive opinion.

Parasitic. Saprophytic micro-organisms are responsible for a great number of cases of vaginitis. To them is attributable the vaginitis not infrequently met after acute exanthematous diseases (with or without desquamation) and in conjunction with divers forms of cutaneous eruptions. The same cause accounts also for the vaginitis observed in strumous and debilitated children suffering from purulent discharges from the nose, ears, etc. Indeed, the number of cases of saprophytic vulvovaginitis would by far exceed all those arising from all other sources collectively were it not for the antagonistic action of the bacillus of Doederlein which normally inhabits the vagina. This vagina bacillus, which is anaërobic and may be cultivated on ordinary media, produces lactic acid during its growth, a quality to which is due the presence of lactic acid in the healthy vagina. In its presence saprophytes, as well as numerous other bacteria, such as the staphylococcus and streptococcus, are unable to develop, and within a short time perish. Gonococci, however, do not yield as promptly to the destructive effect of the vagina bacillus; hence the frequency with which gonorrheal vulvovaginitis is met, notwithstanding the resistance offered to the entrance of gonococci into the vagina by the stratified squamous epithelium lining it.

Saprophytic.

As stated before, contamination of the vagina by criminal assault is comparatively very rare. Much more frequently infection takes place by voluntary sexual act or accidentally. Little girls sleeping with their parents, elder brothers, sisters, or nurses suffering from gonorrhea, may contract the disease by coming in contact with soiled bed-clothes, cotton pads, or other articles used for cleansing purposes.

Gonorrheal.

Gonorrheal vulvovaginitis runs a more or less virulent course, and in hospitals and asylums where many children are congregated in comparatively close quarters, and frequently make common use of infected bathtubs, toilets, etc., the disease is very apt to become epidemic as well as endemic. In one epidemic under my care, in an orphan asylum, comprising over one hundred cases, it required many months of very active treatment to eradicate the affection. Arrest of further spread of the gonorrhea was not effected until every patient was isolated and kept in bed for several weeks. A biweekly examination of every female inmate of the institution (including the nurses in charge) for vaginal discharge was continued for several weeks after disappearance of the last case of vaginitis.

Prophylaxis.

Such procedures form the main prophylactic measures against the disease. Of course, the patients must be restricted from the common use of chambers, bedding, bathtubs, etc. In hospitals and asylums, admitting physicians should be particularly careful to exclude all children having a purulent vaginal discharge, unless provisions be made for the isolation and treatment of such cases. This point is well worthy of consideration, as it would greatly aid in checking further transportation of the disease. As the majority of cases of vulvovaginitis is observed among school-children, a suggestion to the health authorities is, perhaps, in

Prevention of epidemics.

order, viz., to instruct the school inspectors to pay more attention to the detection and isolation of the cases of gonorrhea in children than they do now.

Isolation.

As gonorrhea in adults, that of children presents a marked tendency toward grave complications. Among 148 cases under my care, the following serious complications were observed: Purulent ophthalmia, 7; local peritonitis, 4; proctitis, 3; arthritis, 4; adenitis, 12. Several cases of pyosalpinx, endocarditis, and pleuritis are on record. However, the more familiar one becomes in eradicating it, the less numerous will become the complications and sequelæ in his new cases.

Complications.

After extensive experimenting I found that gonorrheal ophthalmia can best be prevented by frequent cleansing of the genitalia and hands of the patients, and by the employment of a large, tightly fitting vulvar pad. The latter should be changed for a clean one at least every three hours. The child should wear one-piece night-drawers during the night as well as day. The ophthalmia may sometimes be arrested in its incipiency—I succeeded in two cases—by instillation of silver solutions after Credé's method. In view of the unusually rapid progress of the ophthalmia, unfortunately, it is not often that the physician has the opportunity to resort to the prophylactic measures, and nothing else remains but to treat the disease actively and skillfully, and, if not already involved, to endeavor to save the other eye from the dreadful infection.

Prevention of gonorrheal ophthalmia.

Involvement of the uterus and adnexa secondarily to gonorrheal vulvovaginitis in most instances results from injudicious use of douches by forcing the vaginal discharge upward into the uterus, Fallopian tubes, etc. The treatment therefore should not be intrusted to the inexperienced.

Danger of forcible douching.

I believe that I am entitled to the credit of having been the first to call attention (*American Medico-Surgical Bulletin,* May 30, 1896) to the occurrence of gonorrheal proctitis as a complication of vulvovaginitis. The rarity with which this complication is observed, notwithstanding the constant exposure of the anus to the gonorrheal vaginal discharge, would seem to prove the comparative immunity of the skin and mucous membrane of the anus and rectum to gonorrheal infection. Moreover, proctitis usually does not develop until late in the course of the vaginitis, *i.e.,* until the skin of the anus and the adjacent structures has become abraded and denuded by the continued irrita-

Gonorrheal proctitis.

tion of the vaginal discharge, or by scratching for the relief of the not infrequently accompanying intense itching.

The diagnosis of gonorrheal proctitis is rendered positive by the presence of the gonococcus in the mucopurulent stools.

Arthritis.

Like the former complication, arthritis, the so-called gonorrheal rheumatism, also develops late in the course of vulvovaginitis. In the majority of cases the inflammation is limited to one joint, usually that of the knee, and occasionally ends in suppuration and ankylosis.

Inguinal adenitis is quite a frequent complication. The glandular enlargement may increase up to a well-marked bubo. It sometimes suppurates as a result of an additional infection by pus microbes.

Differential diagnosis.

The differential diagnosis between the different varieties of vulvovaginitis can readily be made by bearing in mind the previously mentioned classification. No examination should be considered complete without a very careful microscopic scrutiny of the vaginal discharge. In doubtful cases a culture will settle the diagnosis. Furthermore, it is well to remember that several etiological factors may be operative in the production of the vaginitis in one and the same patient. Hence, the finding of pinworms, for example, in the vagina should not lead us to conclude the absence of gonococci.

Cleanliness.

The treatment of vulvovaginitis varies greatly with the cause. Non-gonorrheal cases usually yield promptly to removal of the etiologic factors (*e.g.,* foreign bodies) and to cleansing of the genitalia with salt, boric acid, or sulphocarbolate of zinc solutions. Gonorrheal vulvovaginitis should be treated by instillation into the vagina (through a soft-rubber catheter) once a day or every other day of half an ounce of a 2 per cent. to 5 per cent. solution of nitrate of silver, followed by neutralization with salt water. After subsidence of the active symptoms douches with mild antiseptics will suffice.

Nitrate of silver.

Tendency to recurrence.

It is well to remember that recurrence of the affection after a period of latency is frequent even under the most careful method of treatment. No case of gonorrheal vulvovaginitis, therefore, should be considered cured unless three or more thorough microscopic examinations of the vaginal discharge prove the absence of gonococci and pus.

MASTURBATION

(Onanism, Thigh-friction).

Production of venereal orgasm by hand, or other unnatural means, is a very common vice among school-children, who usually acquire the vicious habit from older playmates, or erotic governesses, etc.

Thigh friction.

Occasionally masturbation is observed in younger children and even in infants. The latter may be seen to rub their thighs against each other or against the bosom of the nurse, or to exert peculiar rocking motions and fall back in a more or less marked state of exhaustion.

Effects of masturbation.

Suspicious symptoms.

The effects of masturbation vary with the frequency and duration of the habit and the psychical condition of the child. In the majority of cases masturbation produces physical and mental debility, especially depression of spirits, headache, palpitation of the heart and emaciation. In boys we may suspect masturbation by excessive elongation of the penis, in girls by the presence of vulvitis, and often stretching of the hymen. Boys are apt to suffer from nocturnal seminal emission and later also impotence.

Removal of irritation.

Surveillance.

Tonics.

In remedying this evil, it is essential to remove all local sources of irritation, such as phimosis, hypertrophy of the clitoris, pinworms, etc. Infants should be restrained from practising the bad habit by mechanical devices (separation of the thighs, tying of the hands). Older children should be placed under proper surveillance and in suitable spiritual surroundings (change of school; nurses!). The general health should be improved by outdoor exercise, cold shower baths, and by a nutritious but bland diet (no liquors). Bromids are indicated to subdue sexual excitement. Dime novels should be eliminated from the child's reading room.

GANGRENE OF THE GENITALIA.

Diphtheria Vulvæ, Noma Vulvæ.

Drugs; trauma; diphtheria.

Gangrene of the genitalia (vulva, penis, scrotum, etc.) usually develops secondarily to grave local inflammatory processes in the vicinity. More rarely it is primary in nature (after too liberal use of strong antiseptic dressings in open wounds, *e.g.*, carbolic acid gangrene in circumcision; the result of direct violence, *e.g*, stuprum) or occurs in connection with diphtheria, dysentery, typhoid, and similar affections.

Whatever the cause, the prognosis is always very serious, fatal termination usually taking place within about ten days from the onset, unless we succeed in checking the spread of the gangrene by early cauterization or excision of the affected part. Diphtheria antitoxin is deserving of trial.

Cauterization.

Diphtheria antitoxin.

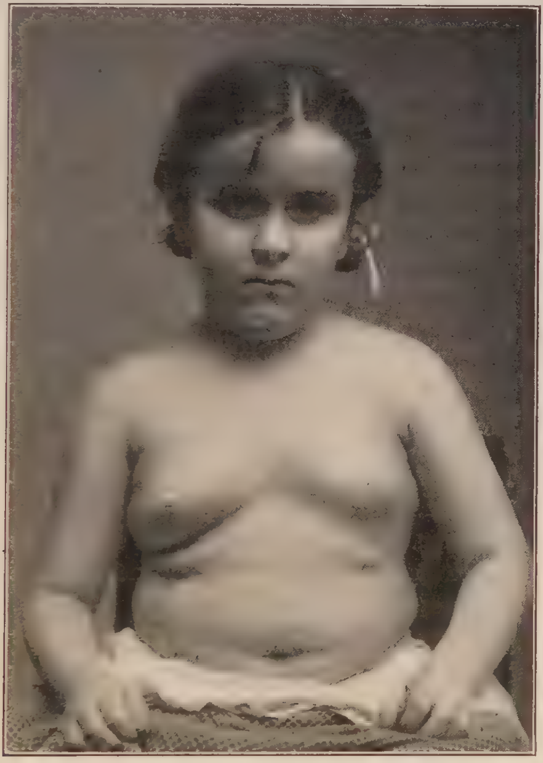

Fig. 143.—Precocity (child 8 years old). (*Sheffield.*)

MENSTRUATIO PRÆCOX.

Genuine precocious menstruation in early childhood is of very rare occurrence. If it does occur, it is usually associated with general bodily and mental overdevelopment. The diagnosis of menstruatio præcox should not be made until vaginal bleeding from local injury, from papillomatous growths, prolapse of the urethral mucous membrane, and hemophilia, has been excluded.

Over-development.

Mistakes in diagnosis.

Precocious menstruation, being free from serious consequences to the general health, calls for no therapeutic measures, except perfect rest during menstruation.

CHAPTER XII.

Diseases of the Blood and Ductless Glands.

Usually secondary.

THE grouping together of the affections of the blood and ductless glands is intended to emphasize their correlation. They are of very common occurrence in children, especially in infancy and in those approaching puberty. At these periods of life, owing to the rapid bodily development, the blood-forming organs are taxed to their greatest capacity, and, hence, are very apt to suffer on slight provocation. The anemias of children are usually secondary in nature, only exceptionally primary. With the present inadequate state of our knowledge, however, no sharp line of demarcation can be drawn between the various types of blood disease. Only too often do we find the clinical and histologic aspects of simple secondary anemia merging into that of splenic anemia, and that of the latter disease into the one of leukemia. The same is true of lymphatic leukemia, chloroma, and lymphosarcoma. For the reasons just stated, therefore, no attempt will here be made to offer an ironclad classification of the diseases in question.

In studying blood disease it is well to bear in mind that the constituents of the normal blood vary within more or less wide limits, and that slight ailments are prone to produce marked disproportion between the number of red and white blood-corpuscles.

Normal blood.

At birth the number of red cells is about 6,000,000, and of white cells, between 20,000 to 30,000 per cubic millimeter. The hemoglobin is very high (about 110 per cent.) and the specific gravity 1066. After the second week the red cells fall to 5,000,000, and the white cells to about 15,000, the hemoglobin to 100 per cent., and the specific gravity to 1050. The red cells are fewer in number in the female than in the male. The *percentage* of the different leucocytes in infants presents the following variations: Polymorphonuclear neutrophiles, 28 to 50; polymorphonuclear eosinophiles, ½ to 10; lymphocytes, 50 to 70, and large mononuclears, 6 to 14. The adult proportion is usually reached by the time the child is six years old. Then the number of leucocytes falls to about 10,000, presenting the following percentage: Neutrophiles, 65 to 75; eosinophiles, ½ to 4; lymphocytes, 20 to 30, and mononuclears, 1 to 4. Normally coagulation of the blood usually occurs within from two to five minutes.

ANEMIA SIMPLEX, CHLOROSIS
(Green Sickness).

Both of these conditions present identical pathologic changes in the blood—reduction in the number of red cells, decrease of hemoglobin, without marked changes in the cells themselves—but differ somewhat in the etiology and course. Thus, while chlorosis is ordinarily encountered in girls at puberty, and almost invariably ends in recovery without any grave alterations in the general health, anemia is a disease of younger children, and if occurring in infants very frequently forms the forerunner of that type of blood disease which is generally described as pseudoleukemia infantum (*q. v.*).

Reduction of red cells and hemoglobin.

Anemia as well as chlorosis is manifested by pallor of the face (green complexion) and mucous membranes, headache, dyspeptic symptoms, undue fatigue after slight exertion, attacks of palpitation of the heart and of dyspnea, general debility and excessive irritability of the nerve system. Auscultation often reveals hemic murmurs along the large veins of the neck and at the base of the heart, which differ from organic murmurs by their inconstancy and frequent change in their intensity and location.

Pallor.

Palpitation of heart.

Debility.

In addition to the aforementioned manifestations chlorosis in mature girls is very prone to give rise to amenorrhea, dysmenorrhea, and less frequently to menorrhagia with consecutive aggravation of the original condition; severe chlorosis is apt to be complicated by venous thrombosis, especially in the lower extremities and the brain sinuses, and occasionally to secondary gangrene and embolism. Of course, such occurrences are very exceptional. The very great majority of cases of chlorosis, as already stated, improve rapidly and fully, although relapses are not uncommon.

Complications.

The management of anemia and chlorosis to a great extent varies with the numerous etiologic factors. The general health should be improved by suitable, nutritious diet, plenty of outdoor air, cold shower baths with gentle massage, ample sleep, and

Nutritious diet.

Fresh air.

Rest.

Iron and arsenic.

avoidance of undue excitement and physical and mental overexertion. Dyspepsia, habitual constipation, diarrhea, loss of blood (epistaxis, etc.), hereditary syphilis, malaria, tuberculosis, heart and kidney affections, and all other diseases as are apt to undermine the system should receive prompt and continuous attention. Where circulatory disturbances are very pronounced, rest in bed is indispensable. Medicinally iron and arsenic are the remedies of choice. The following combination acts splendidly:—

℞ Liquoris arsenici chloridi	ʒj	4
Tincturæ ferri chloridi	ʒiij	12
Syrupi aurantii ....q. s. ad	℥iij	90

M. Sig.: One teaspoonful every three hours for a child 6 years old.

In older children, to avoid destruction of the teeth, the iron and arsenic, without the syrup, may be prescribed with instructions to be taken in capsule form, each dose being prepared before taking it in accordance with the directions given on page 115.

Digestives and tonics (cod-liver oil) will be found to act as useful adjuvants. Change of air, preferably to mountainous regions.

PSEUDOLEUKEMIA LYMPHATICA

(Hodgkin's Disease, Adénie, Lymphadenoma).

Multiple lymphadenitis.

This disease is characterized by multiple hyperplasia of the lymph glands with progressive anemia. The cervical glands are most commonly and severely attacked, but the lymphoid tissue of the entire body is more or less involved. It closely resembles tuberculous adenitis, except that it is much less common than tuberculosis and that in the latter condition the glands show a greater tendency to caseation and suppuration. In doubtful cases the tuberculin test may prove decisive in the diagnosis.

The changes in the blood and the clinical manifestations are identical with those observed in anemia. Occasionally there are local pressure-symptoms, such as pain, edema, cough and dyspnea.

The same as in simple anemia.

Under suitable treatment, which is essentially the same as in simple anemia, recovery or, at least, arrest of the disease is possible. Intractable cases often terminate in leukemia.

PSEUDOLEUKEMIA INFANTUM SPLENICA
(Von Jaksch or Splenic Anemia).

In contrast to genuine leukemia, pseudoleukemia splenica is of quite frequent occurrence, slow in its course and favorable in its outcome. The etiology of this affection is obscure. As a rule, it is observed in connection with pronounced forms of malnutrition, especially rachitis.

Slow course.

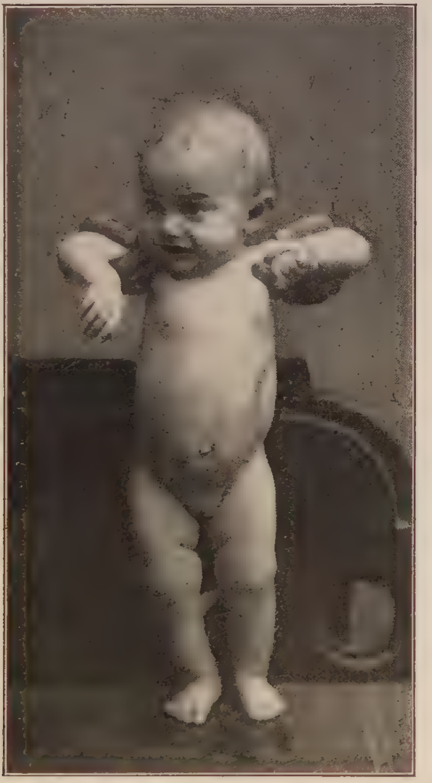

Fig. 144.—Pseudoleukemia Infantum Splenica. Note position of enlarged spleen. (*Sheffield.*)

The chief alterations in the blood are reduction of red cells and hemoglobin, the presence of many nucleated red corpuscles, and an increase in the number of leucocytes, mostly of the mononuclear type. This blood picture essentially corresponds to that of ordinary secondary anemia. In pseudoleukemia infantum,

Increased leucocytosis.

Enlarged spleen.

however, there is marked enlargement of the spleen and occasionally also of the liver and lymphatic glands.

The general symptoms differ but little from those observed in severe anemia. The same applies to the treatment. The syrup of the iodid of iron with the syrup of the hypophosphites seem to exert a specific action in the majority of cases.

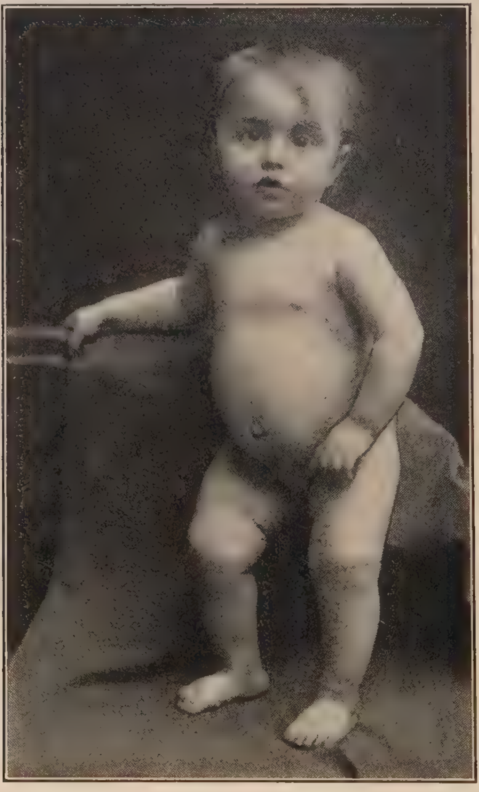

Fig. 145.—Same case as Fig. 144 after three months' tonic treatment. Note reduction in size of spleen. (*Sheffield.*)

LEUKEMIA
(Leucocythemia).

High leucocytosis with unusual types of blood-cells.

As the term indicates, leukemia is characterized principally by an abnormal increase in the number of leucocytes (sometimes reaching as high as a million), and by the presence of unusual types of these cells, *i.e.*, Markzellen (myelocytes), Mastzellen (nutritive cells), and giant basophiles. From a pathologic point of view it is customary to distinguish two forms of leukemia: 1. Lymphatic leukemia, in which the lymphatic glands are chiefly involved (hyperplasia), and 2. Splenomedullary or myelogenic form, in which the spleen (greatly increased in size) and the bone marrow (hyperplasia) are the principal seats of the lesion. Mixed forms also are encountered. The principal difference between the two forms of leukemia are the preponderance of lymphocytes in lymphatic and myelocytes in splenic leukemia. The red cells and hemoglobin are reduced in both varieties.

Lymphatic.

Splenic.

Mixed forms.

The clinical manifestations are essentially identical with those of pernicious anemia, plus enlargement of the lymphatic glands, or spleen and liver. The disease may run a very acute course (acute leukemia), and end fatally within a week or two, or proceed a slower course (chronic leukemia), and lead to a fatal issue after a few months.

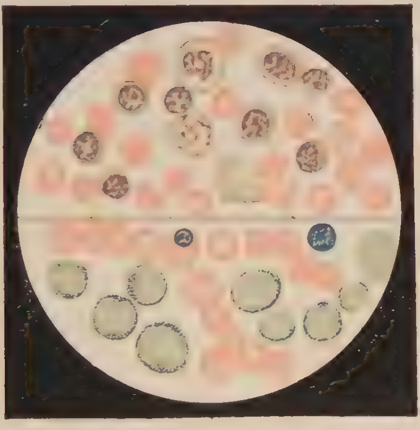

Fig. 146.—Acute Leukemia. This picture is made from two different, rapidly fatal, clinically similar cases. The upper portion is stained with Ehrlich's stain with eosin-hematoxylin; the lower portion is stained with the Plehn-Chenzinsky's stain. (*Lenhartz* and *Brooks.*)

Grave prognosis.

As the nature of leukemia is entirely obscure, little else can be done but treat it symptomatically.

PERNICIOUS ANEMIA.

This form of anemia is characterized by great diminution in the number of red cells (2,000,000 to 1,000,000 per centimeter); reduction in the total quantity of hemoglobin with a comparative

Great reduction of red cells.

Megaloblasts.

Normal leucocytosis.

increase of the hemoglobin in the red cells; increase in the size of the red cells with predominance of megaloblasts; loss of cohesive quality of the red cells (their failure to form rouleaux), and, finally, absence of distinct change (or slight reduction) in the number of the leucocytes.

Intestinal parasites.

This blood affection is very rarely met in children. As in adults, it may occur secondarily to protracted simple anemia or in consequence of abstraction of blood by intestinal parasites, *e.g.*, bothriocephalus latus; uncinaria (*q. v.*).

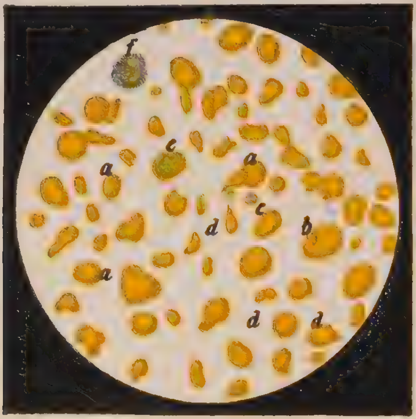

Fig. 147.—Progressive Pernicious Anemia. The case ended fatally in six weeks; cause unknown; possibly in connection with typhoid fever. Ehrlich's triacid stain. Zeiss ocular 1, oil immersion 1/12. *a,* normal erythrocytes; *b,* megalocytes; *c,* microcytes; *d,* marked poikilocytosis; *e,* megaloblast; *f,* polynuclear neutrophilic leucocyte. (*Lenhartz* and *Brooks.*)

Hemorrhages.

In the beginning the symptoms resemble those of severe simple anemia (*q. v.*), but at a later stage of the disease the condition is greatly aggravated by supervening hemorrhages from the mucous membranes, cutaneous ecchymoses and general dropsy. In such cases death invariably occurs within a few months.

Post-mortem examination usually reveals fatty degeneration of the internal organs.

The treatment is the same as in severe anemia (see page 472). Besides, removal of intestinal parasites, if present.

HÆMORRHŒA CONGENITA
(Hemophilia).

Hemophilia is an inherited, congenital tendency to post-traumatic or spontaneous, profuse, often uncontrollable hemorrhage. It affects boys much more frequently than girls and shows a predilection for those of the Hebrew race. The disease becomes less marked with advancing age.

The nature of the disease is still obscure. It is reasonable to suppose, however, that some toxic hemolytic agent transmitted through the spermatozoa or the maternal blood exerts its deleterious influence upon the embryo in its very earliest development, leading to permeability and friability of the blood-vessel walls and lessened coagulability of the blood—these being the only characteristic changes met with in hemophilia with a certain degree of regularity.

Lessened coagulability of the blood.

While, as previously alluded to, the hemorrhage may start spontaneously, in the great majority of cases it follows some trivial injury. A scratch or the prick of a pin or slight abrasion of the body surface, vaccination, snipping of the frenum, circumcision, extraction of a tooth, opening of abscesses, etc., are followed by severe often uncontrollable hemorrhage. Any undue exertion of a muscle or a group of muscles (*e.g.*, jumping off a chair, sudden twisting of an arm), a bump or a blow, etc., often gives rise to a profuse extravasation of blood into the skin or joints. Forcible blowing of the nose may be followed by an exsanguinating nosebleed, and in a case under our observation sneezing produced an enormous hemorrhage from the nose and ear (rupture of the drum!) which nearly ended fatally. In girls hemorrhages may occur from the vagina (often mistaken for menstruatio præcox) long before the age of puberty; and with establishment of menstrual function, the bleeding may be so profuse as to leave the patient monthly in a state of collapse. Hematemesis, hemorrhage from the bowels and hematuria are less common, and bleeding into the serous cavities (peritoneal, pleural and pericardial) and the brain are still less frequent. Hemophilia in the newly born may be manifested during or immediately after birth by severe hemorrhages occurring from abrasions and contusions sustained during delivery, or after cutting the umbilical cord. These hemorrhages are not to be mistaken for hæmorrhœa neonatorum complicating sepsis (see page 181) or the so-called transitory hemophilia which is manifested by idiopathic umbilical

Spontaneous hemorrhage or after trivial injury.

Not to be mistaken for sepsis neonatorum.

hemorrhage (see page 174) or fearful, sometimes fatal bleeding following ritual circumcision. In this form of hemophilia the tendency to hemorrhage is greatest between the seventh and fourteenth days of life, gradually lessening in intensity until the infant reaches the age of two or three months, when it disappears entirely. The differential points of diagnosis between hæmorrhœa congenita and hæmorrhœa acquisita will be spoken of in the discussion of the latter affection (see page 480).

Transitory hemorrhage.

Little can be expected from treatment, except in mild forms of hemophilia ("partial bleeders"). Sterilized, liquid gelatin, (10 per cent.), administered twice daily, for months at a time, per mouth, rectum or hypodermatically has proved very serviceable, especially in partial bleeders. Calcium chloride, in from 2-grain to 5-grain doses, twice daily is useful. Thyroid gland substance, in small doses, continued for weeks at a time, is deserving of trial. To arrest the hemorrhage we may resort to the actual cautery, compression, suprarenal extract, perchlorid of iron, etc. Recently fresh rabbit-serum and horse-serum have been highly recommended.

Partial bleeders.

We should guard against injuries and operative interference (gelatin feeding before operation is helpful) of all kinds.

Prophylaxis.

Bleeders, especially females, should not marry.

HÆMORRHŒA ACQUISITA

(Purpura Simplex. Purpura Hæmorrhagica s. Morbus Maculosus, Purpura Fulminans).

Purpura is an acquired affection of the blood or its vessels characterized by hemorrhages into the skin, mucous membranes and other tissues and more or less marked constitutional disturbance.

The etiology of the disease is still obscure, but is probably a specific micro-organism which invades the blood.

Probably of microbic origin.

Purpura is most frequently observed in children (male and female) over five years of age, and more rarely in younger ones. It occurs either as a primary affection or in connection with acute infectious diseases, such as scarlatina, measles, typhoid, etc., and shows a predilection for poorly nourished, anemic and rachitic children living in dark, damp dwellings, with bad hygienic surroundings.

Free from hemorrhagic tendency at birth or soon thereafter.

Consonant with the degree of severity of the affection, it is customary to distinguish the following forms of purpura:—

1. Purpura Simplex.—The hemorrhage is confined to the skin only, and appears as pinhead- to lentil-sized spots at first upon the lower extremities, but later also on the other portions of the body. Aside from occasional prodromata consisting of gastroenteric disturbance of brief duration, it is free from constitutional manifestations. The majority of these cases pursue a favorable course. The petechiæ either subside entirely within from one week to one month, or return at shorter or longer intervals, in which latter event transition into a severer type of the disease is not uncommon.

Cutaneous hemorrhage.

2. Purpura s. Peliosis Rheumatica. (See page 424.)

3. Purpura Hæmorrhagica (Morbus Maculosus Werlhofii).—This form of purpura is manifested by hemorrhages in the skin as well as in the mucous membranes. Its onset is either sudden or preceded by slight prodromata or purpura simplex. The skin petechiæ may vary in size from a lentil to the palm of a hand, and do not disappear on pressure. They usually spread rapidly over the entire body. The hemorrhages into the mucous membranes are rarely very profuse. As a rule, there are only ecchymoses upon the mucous membranes of the nose, gums, and pharynx, but in severe cases the hemorrhagic tendency may extend to almost every structure and organ of the body, so that the patient bleeds from the nose, mouth, ears, retina and choroid, throat, lungs, stomach, bowels, kidneys, genitalia, etc., and sometimes even into the brain and cord. Under these conditions there are well-marked constitutional symptoms (prostration, headache and articular pain, cerebral symptoms as a result of the anemia or meningeal hemorrhage, colic and tenesmus, etc.), but in mild cases the patient may appear perfectly well. The course of the disease, therefore, varies with the seat and amount of the bleeding. An attack of purpura hæmorrhagica of medium severity usually lasts from ten to fourteen days. After about a week the cutaneous ecchymoses begin to change from the original red to bluish, yellow, greenish and brown, and disappear entirely within another week. The hemorrhages from the mucous membranes and viscera also gradually cease, the general condition of the patient improves, and recovery ensues, apparently without any serious consequences. On the other hand, in a great many cases, not only may the course of the first attack be protracted for weeks and months by frequent recurrences of the bleeding, and lead to profound anemia and death, but a tendency to relapses is

Hemorrhage in the skin and mucous membranes.

Recurrences.

not rarely established, which may manifest itself on slight provocation.

4. **Purpura Fulminans** (Henoch).—This type of purpura is essentially identical with the former variety, except that its course is extremely rapid and violent, with severe constitutional symptoms, such as chills, vomiting, hyperpyrexia, cerebral symptoms, and collapse. It is invariably fatal, death taking place with symptoms of cardiac paralysis, within from one to four days. Post-mortem examination is negative.

Violent course.

Purpura may occasionally be complicated by gangrene of the skin, subcutaneous tissue or mucous membranes, rendering the prognosis very much worse.

Differential diagnosis.

In the early stage of the disease hæmorrhœa acquisita may be mistaken for hæmorrhœa congenita, infantile scurvy, and exanthemata (scarlatina, morbilli diphtheria, variola, typhoid, etc.) with hemorrhagic symptoms.

Hemophilia presents a history of an hereditary tendency, most frequently follows some local injury, and if it occurs spontaneously almost never involves several portions of the body simultaneously.

Infantile scurvy is an affection principally of early infancy and associated with malnutrition. The hemorrhage is also deep-seated (subperiosteal).

Exanthemata have pathognomonic symptoms of their own which are wanting in purpura. The concurrence of the former with the latter, however, should not be lost sight of.

Purpura associated with *sepsis* can readily be recognized by the septic symptoms.

The treatment of purpura is very unsatisfactory. Mild cases usually recover spontaneously, and grave ones may go from bad to worse even under the best mode of treatment. Absolute rest in bed, nutritious diet, plenty of fresh air, iron and arsenic, and the administration of fresh fruit-juice will enhance the arrest of milder forms of the disease.

Treatment.

The hemorrhagic tendency may in some cases be checked by means of suprarenal gland extract, aromatic sulphuric acid, calcium chloride, and spirits of turpentine. Local hemorrhage should be treated in accordance with the rules laid down for the management of bleeding from other causes (compression, ice-bags, styptics, etc). After cessation of the bleeding tonics are useful. Stimulants, in collapse.

MORBUS ADDISONII
(Bronzed Skin).

The pathogenesis of this affection is as yet awaiting correct interpretation. While in the majority of cases post-mortem examination reveals disease of the suprarenals (caseation or calcification), cases of Addison's disease are also on record which failed to show distinct pathologic changes in these glands. The disease usually attacks children over ten years of age and exceptionally younger ones. It is manifested by progressive emaciation, dyspepsia, uncontrollable diarrhea, anemia, and bronze-like discoloration of the skin. The discoloration begins at the breast nipples, axillary regions, hands and face, and gradually affects the entire body. The patients succumb within from a few months or years to exhaustion and paralysis of the heart.

Caseation and calcification of suprarenals.

Hematinics, roborants, and, possibly, suprarenal, parathyroid and pituitary extracts are deserving of trial.

DISEASES OF THE SPLEEN.

Spleen affections are manifested principally by enlargement of the organ—demonstrable by palpation and percussion.

MOVABLE SPLEEN
(Wandering Spleen, Lien Mobilis).

This condition is important chiefly from a diagnostic point of view, as it is apt to be mistaken for splenic enlargement. It differs from the latter by the absence of constitutional symptoms and by the softer consistence of the spleen. It is usually associated with general atony of the entire musculature, especially of the abdominal wall, and in older children not rarely with sinking of the intestines, floating liver and kidneys. Subjective symptoms may be absent. Older children may complain of a feeling of weight or pain in the left side, colic, and nausea.

General muscular atony.

Mild cases frequently obtain permanent relief from the use of an abdominal binder and general tonic treatment (massage, cod-liver oil, arsenic). In very pronounced cases splenectomy is indicated.

ACUTE SPLENITIS
(Splenic Congestion).

An acute splenic enlargement may be caused by malaria, typhoid, recurrent fever and miliary tuberculosis; more rarely by

Usually secondary. influenza, rotheln, scarlet fever, tuberculous meningitis, mumps, erysipelas and angina. Very rapid and intense enlargement of the spleen may occasionally be followed by rupture of the spleen, hemorrhage in the abdominal cavity and death.

In the majority of instances the splenitis subsides spontaneously with the underlying cause. If the disease is due to direct infection by pyogenic micro-organisms, trauma (with open wound) or metastasis, it may end in suppuration (splenic abscess). Occasionally the inflammation extends to the surrounding tissues, especially to the capsule of the organ, *perisplenitis*, and gives rise to inflammatory adhesions to neighboring structures (diaphragm, colon or fundus ventriculi).

CHRONIC INFLAMMATION OF THE SPLEEN

(Chronic Hypertrophy, Splenomegaly).

Occasionally chronic enlargement of the spleen is the result of acute splenitis. Most frequently, however, it develops insidiously in connection with chronic malaria, syphilis, tuberculosis, rachitis, leukemia, pseudoleukemia and amyloid degeneration.

Pressure symptoms. The symptoms vary with the original cause and the degree of pressure exerted by the spleen upon the neighboring structures. No attempt will therefore be made to go into a detailed description of the symptomatology. Mention may here be made of the **Idiopathic form.** fact that in the so-called "idiopathic" splenomegaly the patient may appear entirely free from constitutional manifestations.

The treatment is symptomatic. If the spleen alone is involved and gives rise to grave pressure symptoms, splenectomy may be resorted to.

BANTI'S DISEASE.

This disease is not infrequently observed in children. It is **Splenomegaly. Ascites.** characterized by splenomegaly, anemia, ascites, cirrhosis of the liver, and hemorrhages. Early splenectomy is said to cure the affection. The diagnosis can be made only by exclusion of similar spleen and liver diseases.

DISEASES OF THE THYMUS GLAND.

The thymus gland consists of two lateral lobes coming in close contact along the middle line. It is situated in the anterior portion of the neck and superior mediastinum, extending from the lower border of the thyroid gland to the upper border of the fourth rib. The thymus varies greatly in size and weight. It is about 2½ inches in length, 1½ inches in width (at its lower portion), and a quarter of an inch in thickness. It attains its greatest development (weighing ¾ ounce) between the first and second years, and undergoes rapid degeneration soon after puberty, so that, at the age of twenty, it is a mere vestige of lymphoid tissue and fat. In children under six years of age, light percussion over the superior mediastinum reveals a triangular field of dullness, its base being on a line with the sternoclavicular articulations, and its apex the second rib. It is well to remember, however, that similar dullness is obtained in enlarged bronchial glands.

Normal anatomy.

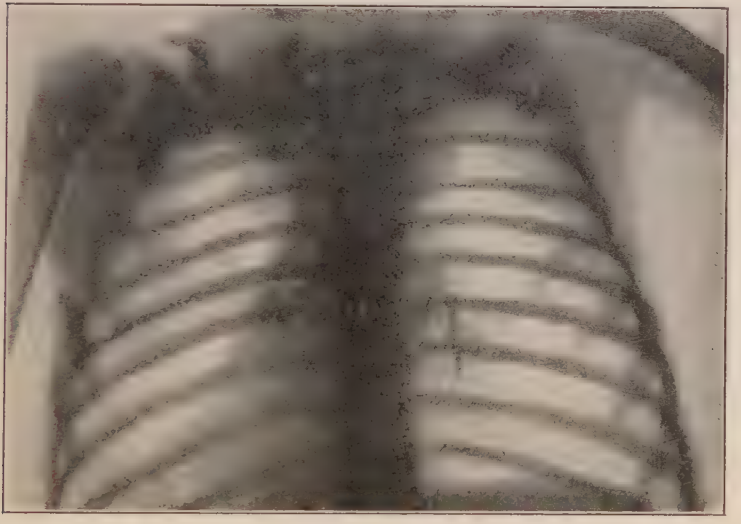

Fig. 148.—Large Thymus (skiagram).

Triangular field of dullness.

Like other glands of the body the thymus gland is subject to acute and chronic inflammation (thymitis) with consecutive hyperplasia, or premature atrophy; tuberculosis; syphilis, and neoplasms.

Thymitis.

Acute thymitis occurs usually secondarily to systemic pyemic processes or by extension of the inflammation from the adjacent structures. The inflammation is very prone to lead to suppuration of the gland.

Hypertrophy of the thymus is sometimes inflammatory in

Hypertrophy.

nature and sometimes of obscure origin. When the enlargement of the gland is pronounced, the diagnosis can frequently be made by an increased area of thymus-dullness over the sternum, by an arched elastic swelling above the incisura sterni, and by its association with enlargement of the lymph glands in the lateral lower region of the neck. The X-rays are often very helpful in the diagnosis. In the majority of instances thymus hypertrophy gives rise to disturbances of the circulatory and respiratory ("asthma thymicum," "inspiratory stridor of sucklings") organs, as a result of pressure upon neighboring blood-vessels, nerves, and trachea.

Dullness; swelling.

Pressure symptoms.

Tuberculosis.

Tuberculosis of the thymus gland, as a rule, is not discovered until post mortem, and hence is of no clinical importance. To a great extent the same is true of syphilis of the thymus, which, by the way, is of very rare occurrence. Both of these pathologic processes usually lead to atrophy.

Syphilis.

Sarcoma.

Sarcoma is the most common new growth of the thymus gland. The symptoms and diagnosis are essentially identical with those of hyperplasia, already referred to.

As the affections of the thymus at best escape detection until a late stage, little can be accomplished in the way of treatment. Where syphilis is suspected the iodids will prove efficient and in cases of neoplasm an attempt may be made to extirpate it. Acute inflammatory symptoms should be relieved by antiphlogistic measures (ice, cupping, and the like).

STATUS LYMPHATICUS

(Thymus Death).

Sudden death after trivial cause.

In a certain number of apparently healthy children sudden death occurs after some trivial cause, such as shock following slight trauma, or operation, injection of serum, etc., inhalation of a minute quantity of an anesthetic and the like. Post-mortem examination reveals no definite lesion in any organ to account for the sudden death, except general enlargement of the lymphoid tissue of the body (adenoids and tonsils, the follicles of the intestinal walls, the peripheral glands, etc.) and especially of the thymus gland. This pathologic condition is being generally described as "status lymphaticus." The pathogenesis of this anomaly is still shrouded in mystery. The view previously held that such deaths were the immediate result of compression of the neighboring vital structures by an enlarged thymus ("thymus death") has been found devoid of indisputable pathologic or clinical foundation.

Whatever the etiologic basis, however, the mere fact that sudden death may follow any of the aforementioned seemingly harmless therapeutic measures should serve as a warning against their employment in children in whom status lymphaticus is suspected. Children suffering from scrofula, rachitis, spasmus glottidis, and the like, belong to this category.

Caution in operating.

DISEASES OF THE THYROID GLAND.

The normal thyroid gland is somewhat larger in children, especially girls, than in adults. It consists of three lobes, one middle small lobe (inconstant) and two larger lateral lobes. The latter are connected by an isthmus. The lateral lobes are situated on each side of the trachea along the second and third tracheal rings; the middle lobe lies in front of the thyroid cartilage and ascends upward in the direction of the middle of the hyoid bone. As the gland is thin and often lies deeply imbedded into the neck, it is very rarely possible to determine the size of a normal thyroid by palpation.

Normal thyroid.

THYROIDITIS
(Strumitis).

Primary inflammation of the thyroid gland is usually of traumatic origin (direct violence, or injury during delivery). It is of very rare occurrence. More frequently we meet with secondary thyroiditis, as a rule, in connection with acute exanthematous diseases and occasionally with parotitis, malaria, and articular rheumatism.

The symptomatology consists of swelling of the gland, pain on pressure as well as on moving the neck, and in some cases redness, fluctuation and suppuration, and more or less marked pressure symptoms.

Swelling, pain, and pressure symptoms.

The inflammation usually disappears under local application of cold. Should an abscess form, it demands immediate evacuation of the pus and drainage.

Severe protracted thyroiditis not rarely leads to atrophy of the gland.

GOITER
(Struma).

Hyperplasia. Cystic degeneration.

As in adults, the thyroid gland of children is subject to hyperplasia and cystic degeneration. In countries where goiter is endemic it is not rarely observed in very young infants, and is probably of antenatal origin. On the other hand sporadic goiter, as a rule, develops at the period of puberty, particularly in girls.

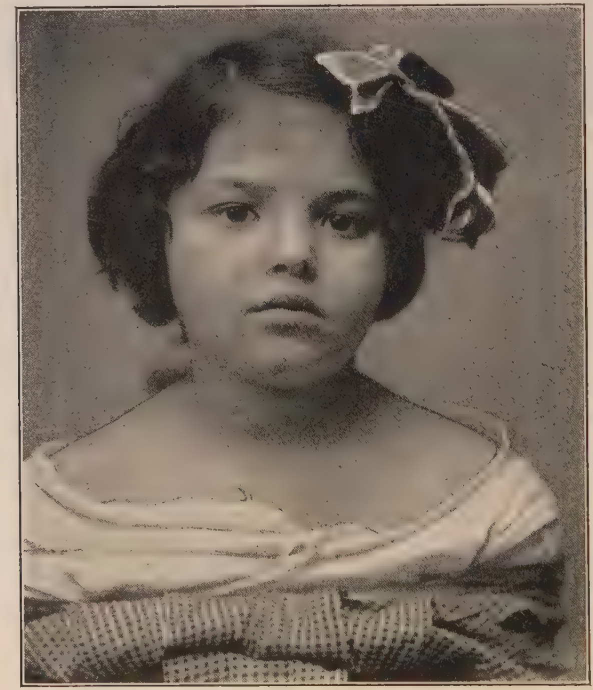

Fig. 149.—Goiter (11 years old). (*Sheffield.*)

Pressure symptoms.

Small goiters may remain free from any manifestations, except the local swelling in the anterior portion of the neck, while goiters large enough to exert pressure upon the adjacent structures may prove a menace to life by compression of the trachea, and the large blood-vessels and nerves which abound in the neck. The pressure symptoms ordinarily consist of headache, dizziness, aphonia, dyspnea and paroxysmal cough. This grave symptom-complex, however, is of unusual occurrence.

On the whole, the prognosis is favorable. The great majority of cases of goiter yield promptly to internal administration of small doses of iodin, with or without thyroid or parathyroid gland substance, and external use of iodin ointment. Large goiters causing marked pressure symptoms call for their extirpation.

In countries where goiter is endemic its development to a great extent may be prevented by change of residence, by boiling the drinking water, and by drinking large quantities of distilled water.

Distilled water as a prophylactic.

In infants goiter may be mistaken for a large hygroma cysticum coli congenitum or other cysts of the neck, and in older children for exophthalmic goiter. Cysts of the neck are characterized by marked fluctuation and rapid development; and usually arise from the submaxillary region.

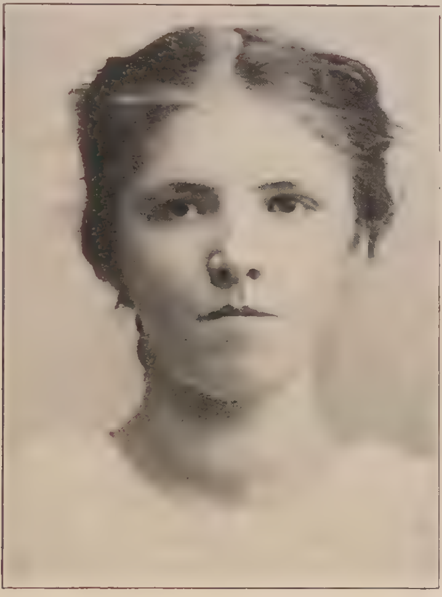

Fig. 150.—Cystic Goiter. Within the last two years patient (13 years old) has been gradually becoming feeble-minded. (*Sheffield.*)

Differentiation from cysts in the neck.

Exophthalmic Goiter (Basedow's or Graves's Disease) is characterized, in addition to the goiter, by tachycardia, muscular tremor, exophthalmos, general ill health, vasomotor disturbances (flushes of the skin alternating with pallor), and pigmentation of the skin.

Tachycardia tremor and exophthalmos.

CRETINISM

(Endemic or Goitrous Cretinism, Sporadic Cretinism or Myxidiocy).

Arrest of thyroid secretion.

Cretinism is due to partial or total arrest of the secretion of the thyroid gland, in consequence of congenital or acquired (extirpation) absence, atrophy (from strumitis, syphilis, tuberculosis, or neoplasms), or goitrous degeneration of the gland.

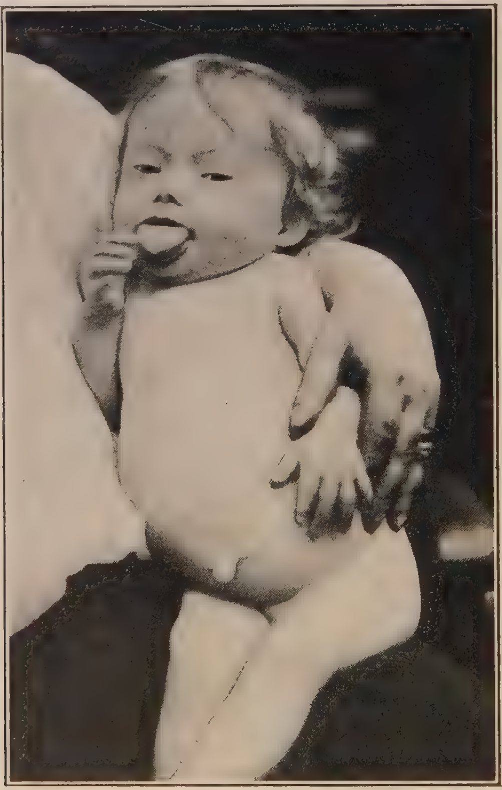

Fig. 151.—Congenital Cretinism. Child 6 months old; showed typical symptoms soon after birth. (*Sheffield.*)

Endemic.

Endemic cretinism occurs in children living in countries where goiter is endemic, or in descendants of people coming from these regions, and is very frequently associated with goiter. On the other hand, sporadic cretinism is observed in children coming from other parts of the world. The term "myxidiocy" is usually

Sporadic.

reserved for the pronounced forms of cretinism which are associated with marked pseudolipomatosis.

Usually congenital.

The great majority of cases of cretinism in children are of antenatal origin, although the pathognomonic manifestations with few exceptions (see Fig. 151) do not appear until the child is over one year of age. At about this time it is usually noticed that the infant's bodily development is arrested, and its intelligence, instead of rapidly progressing, grows perceptibly backward. The fontanelles remain open; the head is large, flat and plump and set upon a thick and short neck. The forehead is low and the root of the nose is broad. The face is weak and senile; the eyelids, lips and tongue are thick and the latter slightly or markedly protrudes from the half-open mouth. The teeth are slow in coming and rapid in decaying. The abdomen is greatly

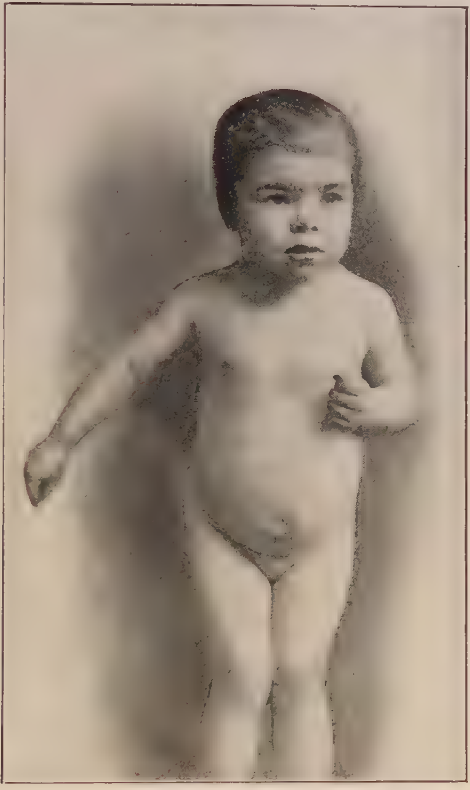

Fig. 152.—Sporadic Cretinism in a Girl 8 Years Old. She measured 33 inches in height. (*Sheffield.*)

Arrest of physical and mental development.

Feeble-mindedness up to total idiocy.

distended, often presenting an umbilical hernia. The extremities are more or less deformed and the articulations thickened. The hands and feet are short, broad and thick. Cretins slowly learn to walk, but their gait is dragging and awkward, with a tendency to fall forward. If left untreated typical cretins rarely attain 3 feet in height. Ordinarily cretins of ten or twelve years of age appear but two years old in stature, and still younger in their mental development. The intelligence of the cretin is invariably below par; it varies, however, greatly with the functionating capacity of the thyroid gland, and the period at which the morbid process makes itself felt. Thus, some cretins ("half-cretins") possess a fair measure of intelligence, appreciate their surroundings and are able to acquire a vocabulary ample to make their wants understood or even to hold an intelligent conversation; others are totally idiotic and grow more stupid with advance in years. The special senses suffer greatly. Taste and smell are obtuse; hearing is defective and not rarely associated with mutism. The voice is husky. In early infancy, before the degree of intelligence is determinable, the diagnosis of cretinism can fre-

Half-cretin.

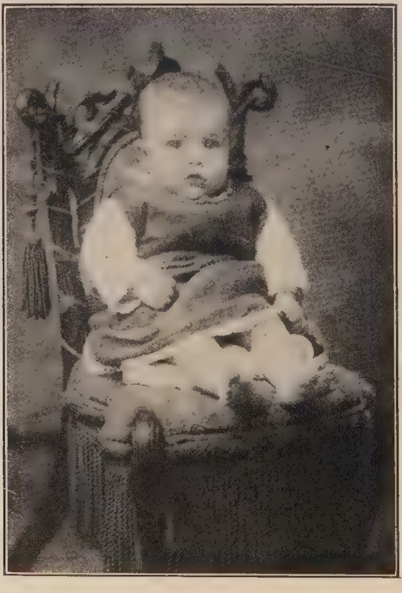

Fig. 153.—Same case as Fig. 152 at the age of one year. Apparently perfectly normal. (*Sheffield.*)

quently be made by the dryness and waxy color of the skin; the profound anemia; the sparseness and brittleness of the hair; the subnormal temperature and the presence of so-called "fatty tumors" in the clavicular regions.

Pseudo-lipomatosis.

As the child grows older it is noticed also that the genitalia and their functions remain in a primitive state. Typical cretins, fortunately, have no power of perpetuating their monstrous kind.

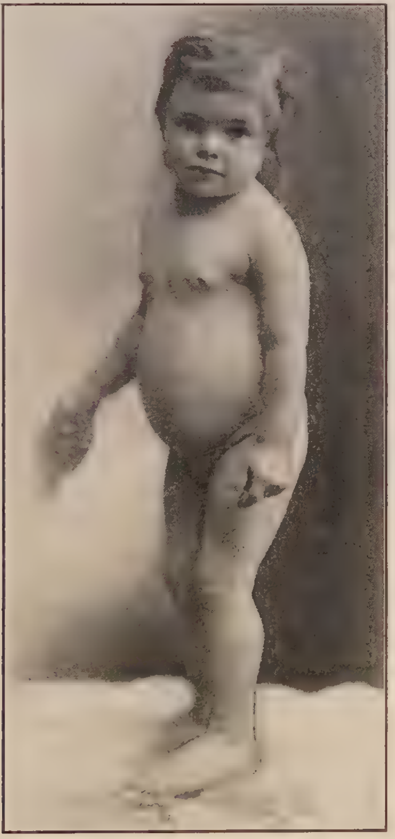

Fig. 154.—Sporadic Cretinism. Same case as Fig. 152, after two months' treatment with thyroid. Note complete transformation of features, etc. She gained 2 inches in height. (*Sheffield.*)

Hopeless without thyroid.

Up to the discovery of the underlying pathologic basis of the disease, cretins used to go on from bad to worse until finally relieved of their miserable existence by death, at an age of from thirty to forty or earlier in consequence of intercurrent diseases. Nowadays, however, a great deal can be done to ameliorate their condition by the administration of thyroid gland. Partial cretins particularly can now be improved sufficiently as to enable them to pursue some occupation and to provide for their maintenance. The results obtained from thyroid medication are often miraculous. After exhibition of thyroid but for a short time—sometimes only a few weeks (see Fig. 154)—the cretin is frequently transformed from an uncouth, apathetic and clumsy little creature into a lusty, gracile and growing human being. The blurred facial features gain youthful expression, the lusterless and withered hair takes on new life, the stunted stature approaches normal proportions and the brutal stupidity slowly gives way to human intelligence. The sooner the treatment is begun and the longer it is persisted in the more certain are the favorable results. At best, however, a cretin always remains childish for life—mentally as well as physically.

Specific effect of thyroid.

The thyroid gland may be administered in the form of extract (1 grain for every year of the child's age, twice a day), the fresh thyroid gland, or any of the numerous thyroid preparations on the market. The effect of thyroid is often enhanced by combining it with parathyroid. (See page 121.)

Mode of administration.

Endemic cretinism may frequently be prevented by treating the pregnant mother with thyroid-gland extract.

Prophylaxis.

For the differential diagnosis between cretinism and other forms of idiocy, the reader is referred to the chapter on "Idiocy and the Allied Mental Deficiencies," page 570. Mention will here be made of the fact that in doubtful cases the diagnosis can often be decided by the experimental administration of thyroid gland.

Differential diagnosis.

The advisability (and success) of transplanting the thyroid gland from a sheep is still subject to controversy.

CHAPTER XIII.

Disturbances of Metabolism.

MARASMUS; ATHREPSIA; INFANTILE ATROPHY (PEDATROPHY).

Intestinal autointoxication.

The nature of this appalling infantile wasting is still shrouded in mystery. It is apparently only a functional disorder, a form of intestinal autointoxication, arising from non-assimilation of the food consumed, since the post-mortem lesions (atrophic patches in some portions of the intestinal tract) are not uniform and rapidly disappear when the atrophic infant is put on a suitable diet, which may vary from an ideal breast milk to some proprietary artificial food (!). In this group, of course, are not included the cases of marasmus accompanying tuberculosis, syphilis and the like.

Apparently normal at birth.

Whatever the pathology and cause, the symptomatology is very pathognomonic. The apparently normally born infant, after thriving fairly well on the milk-mixture it has been receiving, begins to show signs of ill health and rapidly loses in weight. The food disagrees; it is vomited or regurgitated. The stools are green and frequent, scanty in quantity, and contain undigested particles of food. The child suffers from colic, especially soon after feeding; is very restless, cries and whines pitifully, sleeps poorly, and, do what you will, the emaciation continues at a rapid pace. Before long the fontanelles, the eyes and cheeks are sunken; the nose and chin pointed; the abdomen is at first prominent but later retracted; the skin wrinkled, often hanging in folds, and adding to this the earthy pallor and senile expression of the face, the poor creature is a sight dreadful to behold. Though dried up to mere skin and bone, with respiration shallow and pulse bad, it keeps on fighting for life for weeks and months—not rarely successfully.

Senile face; shrunken body.

Unless wrecked by intercurrent diseases, those showing tenacity to life, and coming under observation not entirely in a hopeless state, stand some chance to recuperate their vitality and to recover completely. The prognosis depends also upon the duration of the marasmus, the age of the patient—it is more favorable in infants over four or five months than in younger ones—and the care it can receive from those in attendance. The concurrence of complications or sequelæ, such as atelectasis, edema, pneumonia, colicystitis, pyelonephritis, ostitis, general furunculosis and the like, greatly mar the chances of recovery.

Complications.

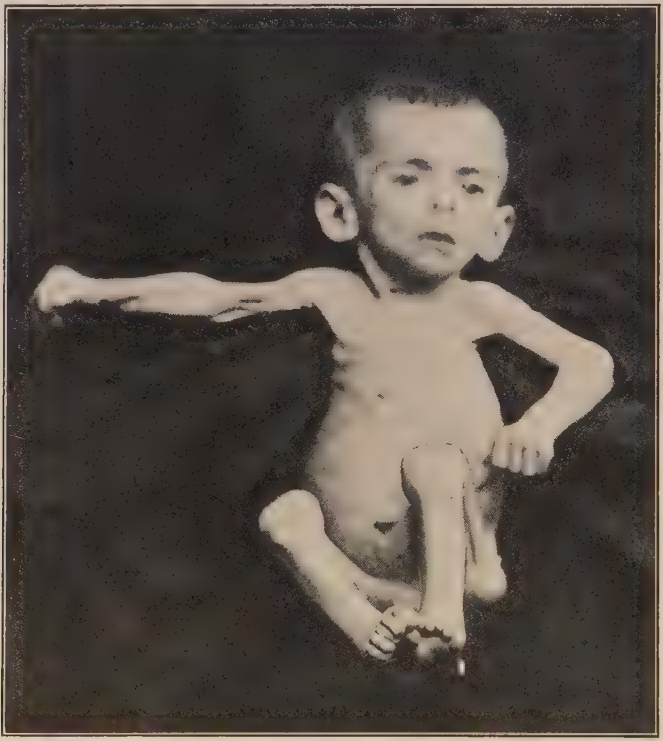

Fig. 155.—Marasmus in a child ten months old. Note "senile face." (*Sheffield.*)

As athrepsia almost invariably occurs in artificially fed infants, the line of treatment which at once suggests itself is to supplant the artificial food by human milk. Indeed, through such a change miraculous improvement in the infant's condition may often be observed within a very few days, requiring no further treatment to complete prompt and uneventful recovery. Wet-nursing, therefore, should be the treatment of choice, even if it be only for a month or two, after which period cows' milk feeding may

Breast milk the best remedy.

frequently, successfully be resumed. Occasionally breast milk does not quite agree at first; but after persistent effort by allowing the baby to nurse only from five to ten minutes at a time, and giving it a little plain-, lime-, or barley-water before each feeding, the difficulty will readily be surmounted. Lavage and colon flushing often act very beneficially. When the services of a wet-nurse are not obtainable (for financial or other reasons), an attempt should be made to feed the baby on "laboratory" milk, always beginning with small quantities of weak milk-mixtures, and gradually increasing in quantity and quality according to indications. Not infrequently whey-mixtures act kindly. With poor people unable to carry out any of the aforementioned suggestions, we may try—for want of or as a stepping-stone to something better—condensed milk in mild dilution with plain- or barley-water (ʒss condensed milk to ℥ij barley-water). Indeed, condensed milk is often invaluable during the summer months, and, if found to agree with the child, should unhesitatingly be continued until cold weather will allow a change for cows' milk. I cannot pass the subject without emphasizing the fact that on a few occasions fearfully marasmic babies were rescued from imminent destruction by means of proprietary infant foods. As this signal success was attained after many other methods of feeding had utterly failed, I am looking upon it as more than a mere coincidence. In some cases "malt-soup" (*q. v.*) acts exceedingly well.

Laboratory milk.

Artificial foods.

Malt soup.

Lavage and colon irrigation are useful in all cases. The latter should be employed daily; the former every alternate day, or more often if the return-water contains large quantities of mucus, and the vomiting persists. In the latter event it is often of advantage to add a little boric acid or bicarbonate of soda to the sterile water used for stomach washing. Of medicinal agents, in addition to an occasional dose of calomel, pancreatin is the only remedy I place some reliance upon. One or 2 grains each of pancreatin and bicarbonate of soda may be administered after feeding.

Lavage.

The mouth of the infant should be kept scrupulously clean, and the buttocks dry and clean—to prevent stomatitis and intertrigo, both of which form common complications. The child should not be left too long in recumbent posture, lest decubitus or passive pulmonary congestion supervene. For details of treatment of atelectasis, edema, and other complications the reader is referred to the respective chapters.

Frequent change of posture.

Outdoor life.

Outdoor life and plenty of fresh air while the patient is indoors are essential to successful management of the cases in question. Whenever possible the child should summer in the country. Above all, however, breast milk is the specific for marasmus, in the way of prophylaxis as well as cure.

See also "Tuberculosis," and "Syphilis."

RACHITIS

(Rickets, The English Disease).

Rickets is one of the most common affections of early childhood. It prevails to a greater or less extent in almost all parts of the world, but shows a predilection for poorly born, poorly nourished (also among the well to do) and poorly housed children of temperate zones. The immediate cause of rickets is an as yet undiscovered micro-organism or toxic product (parathyroid disease?) circulating in the blood. As its direct and most conspicuous result we have great diminution of the inorganic elements of bones, exaggerated production of epiphyseal cartilage, excessive cell proliferation beneath the periosteum, and incomplete ossification of the new osseous tissue. As the disease advances chronic inflammatory changes occur also in the different soft structures (muscles, arteries, etc.) and organs (spleen, liver, etc.) of the body, leading to a complex pathologic entity *sui generis*—entirely distinct from any other diseased process.

Deficiency in lime salts.

This pathogenic process is very insidious in its onset and its course; hence in the beginning rickets is very apt to be overlooked, especially if following upon some other illness.

As a rule, the initial symptoms are very vague, and consist of recurrent indigestion, restlessness and debility—a non-pathognomonic group of symptoms rarely arousing the anxiety of those in charge of the patient as to seek medical advice. When seen by the physician, therefore, the disease is usually in full bloom.

Soft, large skull; open fontanelles, and baldness.

The skull is relatively large, the forehead broad and prominent in profile (frons quadrata). The parietal eminences project strongly, and the fontanelles, especially the anterior one, and the sutures fail to close in due time. The occiput is thinly covered by hair or entirely bald, and here and there yields to pressure with the finger (craniotabes).

The local baldness is the result of undue pressure and friction of the occiput against the pillow, and the effect of profuse perspiration which is most marked at the posterior portion of the head. The sweating and rubbing of the head, both very early symptoms of rickets, in a way are correlated, and probably due to cranial hyperemia.

Deformed jaw. The lower jaw instead of being rounded becomes flattened, and its alveolar edge turned inward. The upper jaw also is more or less deformed, and the teeth, which are late and irregular in coming, are asymmetrically set, conforming with the altered shape of the jaws.

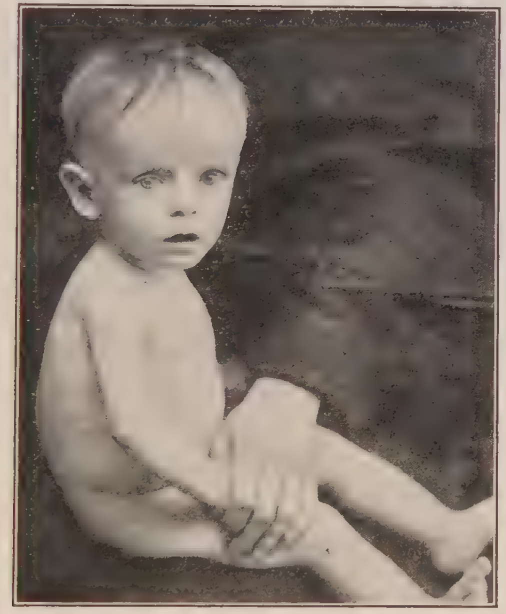

Fig. 156.—Rachitic Frons Quadrata and Curvature of Spine. (*Sheffield.*)

Faulty teething. Owing to the deficiency in enamel the teeth soon turn yellow, brownish or black, are streaked and brittle and subject to rapid decay.

Deformed thorax. The rachitic thorax is very typical in appearance. The clavicles are more sharply curved than in the normal, and occasionally infracted; the costochondral junctions are thickened, bead-like in shape (most marked from the fourth to the eighth rib), assuming in their sloping course from above downward a **Rosary.** rosary-like appearance (*rachitic rosary*); the sides of the thorax are flattened and the sternum projects, as in birds,—hence

Pigeon- or chicken-breast. the so-called "pigeon-" or "chicken-" breast (*pectus carinatum*), and, finally, the lower lateral diameter is widened.

The vertebral column, though rarely affected in mild forms of rachitis, invariably suffers in severe and protracted cases. The deformities most frequently met with are kyphosis and scoliosis.

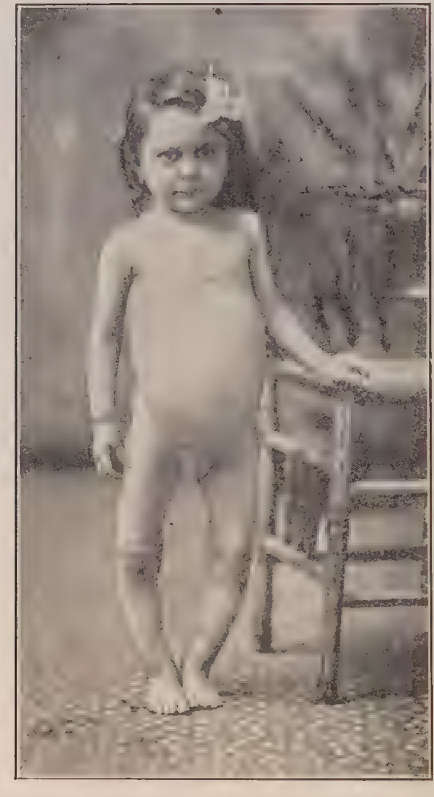

Fig. 157.—Rachitic Beading of Ribs, "Pot-belly" and Bowlegs. (*Sheffield.*)

Kyphosis. The kyphosis or backward curvature usually extends from the middorsal to the sacral region. It differs from tuberculous kyphosis by being rounded, and in the early stages reducible when the child is placed upon the abdomen and the thighs are overextended (see "Spondylitis," page 381). Differentiation from spondylitis. Rachitic lateral curvature or scoliosis is produced by the relatively heavy weight of the head upon the yielding (muscular and ligamentous insufficiency) vertebral column. The condition is further aggravated by allowing the patient to sit up or walk at too early an age and for too long periods and by the habitual unequal distribution of the encumbrance.) As regards the latter it will be noted that right-handed persons usually carry their children on the left arm, so as to have the right hand free, and, in consequence, the right pelvis of the child is lifted upward, the right shoulder tilted downward and the middle spine shoved laterally—lateral scoliosis with the spinal convexity to the left. While rachitic scoliosis is most frequently observed in early childhood, rickets undoubtedly forms also the principal cause of the so-called postural scoliosis of school-children, the curvature being merely an exaggeration of the former condition. Rachitic scoliosis is to be differentiated from congenital scoliosis (very rare; as a rule associated with other congenital deformities); cicatricial scoliosis (following operation for purulent pleurisy); paralytic scoliosis—in association with poliomyelitis, etc. (see Fig. 114); spondylitic scoliosis—usually kyphoscoliosis (see "Spondylitis," page 381);

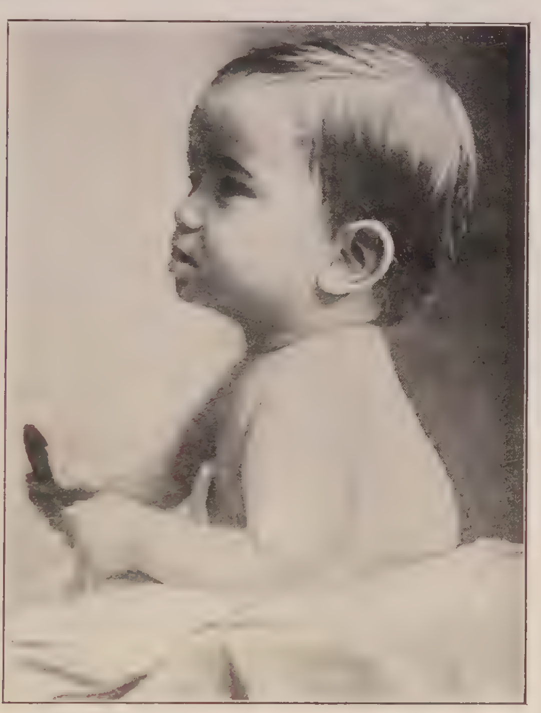

Fig. 158.—Rachitic Kyphosis in a Boy 20 Months Old. Note superabundance of fat. (*Sheffield.*)

Scoliosis.

Differentiation from other varieties of scoliosis.

static scoliosis (in congenital or acquired shortening of one lower extremity). Although, as previously alluded to, rachitic scoliosis is reducible in its early stage, if left alone for a long period the deformity is apt to remain permanent, notwithstanding the disappearance of the other symptoms of rachitis.

Epiphyseal enlargement.

The extremities very rarely escape involvement. In the upper extremities we usually find marked enlargement of the epiphyses at the wrists, and less frequently at the elbow. In creeping infants the radius and ulna are often curved and sometimes infracted, and in severe cases the hand is separated as it were by a furrow—"double jointed." Occasionally there is also an enlargement of the ends of the metacarpal bones or the phalanges.

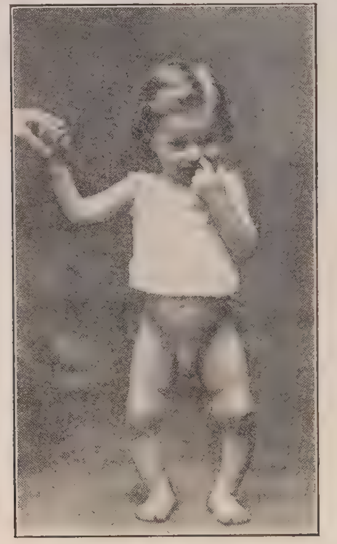

Fig. 159.—Rachitic Bowlegs, "Jug"-shaped Abdomen, and Separation of Epiphyses — "Double-jointed." (*Sheffield.*)

Curvatures of lower extremities.

By far more marked are the deformities of the lower extremities. The soft tibia and fibula are ill prepared to balance the weight of the body. The flimsy fundament thus tumbles under its encumbrance. The hapless patient learns to walk late and with difficulty, or, as it were, "forgets" or unlearns how to walk. If he continues to walk, the tibia and fibula bend either outward (bowlegs — genu varum; O-shape), inward (knock-knees—genu valgum; X-shape), forward (saber-blade shape), or in severe cases simultaneously in different directions. As in the upper extremities there is also an enlargement of the epiphyseal ends of the bones, and occasionally infraction of the diaphyses. Children sitting crossed-legged may present also more or less pronounced curvatures of the femur and pelvis. Rachitic flatfoot is rare.

The course of these deformities varies. In the majority of mild and moderately severe cases spontaneous recovery occurs with improvement of general condition. On the other hand, in extreme cases, where, as a rule, growth is greatly retarded, the curvatures persist and require forcible corrective measures.

The muscles generally participate in the rachitic process. They are thin and flabby and partly responsible for the difficulty in sitting and walking ("pseudoparalysis"), abdominal distention ("pot-belly"), and for the constipation and prolapsus recti. The muscular insufficiency may be associated with overfatness (see Fig. 158) and mask the local rachitic manifestations.

Pseudo-paralysis.

Pot-belly.

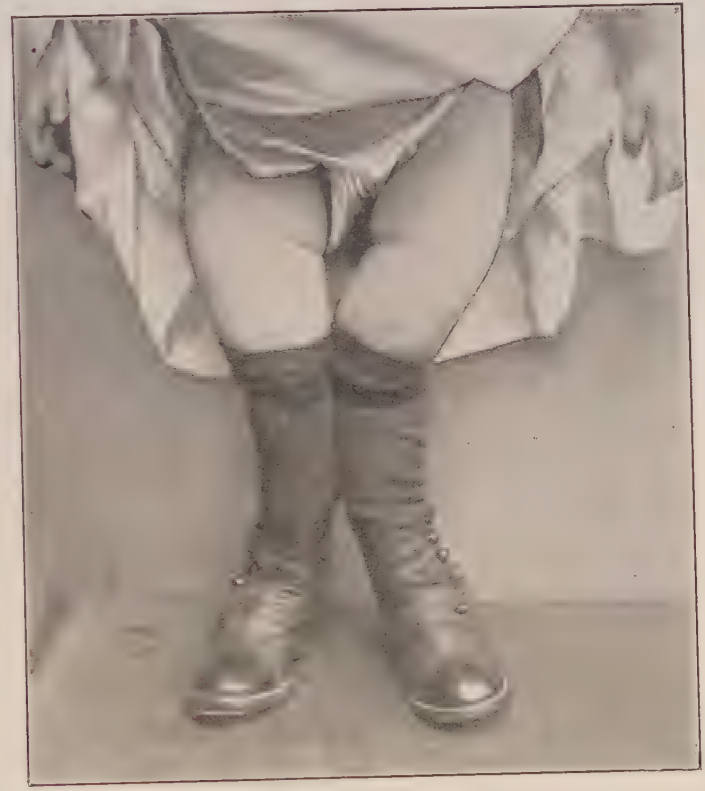

Fig. 160.—Rachitic Knock-knees. Note also infraction of left femur. (*Sheffield.*)

The ligaments are more or less lax allowing undue mobility at the larger joints, and giving rise to the abnormality known as "double-joints."

Double-jointed.

Coincidently with and in a measure because of the gross alterations in the body framework manifold changes occur also in the functions and structures of other components of the body.

Respiratory difficulties.

Anemia.

Spasmodic affections.

The respiratory system suffers early. The contracted chest compresses its contents and disturbs the equilibrium of the thoracic and abdominal organs. The area of breathing space is reduced, hence, respiration more or less interfered with, and the tendency to respiratory disease greatly increased. The latter is favored also by the timidity of the parents to expose their delicate babies to outdoor air, keeping them huddled up in poorly ventilated rooms and thus reduce their power of resistance to infection. In consequence of it slight catarrhs of the nasopharynx or larynx instead of, as in the normal, yielding promptly to suitable treatment, persist indefinitely and lead to capillary bronchitis or bronchopneumonia, not rarely with fatal issue or greatly protracted convalescence with a predisposition to tuberculous infection. As an immediate result we have also profound secondary anemia—reduction of hemoglobin and red blood-cells and moderate leucocytosis. The child is pale, sometimes waxy in color; its digestion is poor; diarrhea alternates with constipation, the latter, however, preponderating. The liver and spleen are more or less enlarged and help to distend the abdomen. Rachitic children are very irritable, sleep restlessly, and show a great disposition toward different spasmodic conditions. Spasmus glottidis, eclampsia and tetany are frequent complications of severe and protracted cases of rickets, especially in very young infants.

Differential diagnosis.

Cases of rickets presenting the local and general symptoms here depicted usually offer no diagnostic difficulties. Less typical cases, however, may be confounded with cretinism, achondroplasia, congenital syphilis, incipient hydrocephalus, and osteogenesis imperfecta—a group of diseases which not only have several symptoms in common and are to a certain extent etiologically correlated, but may also be associated with rickets.

In *cretinism* there is marked mental deficiency; the tongue is thick and protruding from the mouth; as the child grows older there is very pronounced disparity between its age and body length.

Achondroplasia is characterized by a striking disproportion between the length of the trunk and extremities; the curvature of the shafts of the bones is due, not as in rickets, to softness of the bones, but to embryonic defective development; the fingers do not lie parallel as in the normal, but are spread out like ribs of an open fan.

The epiphyseal thickening at the ribs and the long bones of *syphilis hereditaria,* as a rule, is observed soon after birth in association with other symptoms of syphilis which yield promptly to specific treatment.

Incipient hydrocephalus has several symptoms in common with rickets (separation of the fontanelles, softening of the cranial bones, irritability of the nerve system). In hydrocephalus, however, the cranial distention is rapidly progressive in character, leaving the long bones of the body, which suffer most in rickets, almost unmolested.

Osteogenesis imperfecta differs from rickets in that in the former the bones are so soft that they can be cut and bent, splintered and fractured in several places.

The importance of an early diagnosis cannot too strongly be emphasized, as upon it depends the prognosis, the success of treatment. While it is generally admitted that rachitis *per se* is not dangerous to life, and that in a number of cases spontaneous recovery is possible, the indifference of the laity as well as the physician regarding early and persistent treatment is strongly to be deprecated. Spontaneous recovery is rarely complete. On the contrary, without suitable treatment the majority of children are left stunted in growth, distorted in shape and features, and depressed in spirit—in short poorly qualified to struggle for an existence and to compete with their fellowmen favored by good fortune with sound mind and body.

Retarded bodily development.

Preventive measures.

Rickets is preventable by abundance of sunlight and fresh air and by a mixed, nutritious diet. In the absence of contraindications, children over three months of age should receive in addition to milk small quantities of carbohydrates; those over six months also thin soups and orange-juice; those over nine months half of a soft-boiled egg, some beef-juice, and a little toasted bread with sweet butter, and those over a year one egg daily, some fresh vegetable soup, oatmeal gruel, light cocoa, etc., and occasionally a small quantity of finely scraped fresh beef (see page 81). Season permitting, raw milk should be given in preference to boiled, sterilized or pasteurized.

Diet.

Rest.

Rachitic deformities may be prevented by avoiding superencumbrance of the spine and extremities. Infants with incipient rickets should, as much as possible, be kept off their feet, and advantageously held in recumbent posture, allowing them to remain in upright position only for short periods at a time.

Sunshine.
Nitrogenous diet.

The suggestions just made apply as well to the management of further advanced cases of rickets. Here, too, sunshine and nitrogenous diet in abundance and removal of the superincumbent weight of the body are the remedies *par excellence.* To these we should add hydrotherapy (sea-salt baths), massage and passive motion, and corrective, light braces where the deformities persist. Operative corrective procedures should be reserved for deformities of over three years' standing, as slight curvatures usually respond to non-operative antirachitic measures.

Hypophosphites.

As auxiliaries, especially with the view of overcoming the anemia and the deficiency of mineral elements, the syrupus hypophosphitum compositus (U. S. P.) and cod-liver oil are of undoubted therapeutic value. Syrupus ferri iodidi with syrupus calcii et sodii hypophosphitum (N. F.) also is of service.

Organotherapy.

In intractable cases organotherapy, especially the extracts of thyroid, thymus and pituitary glands and red bone marrow should be given fair trial. A sojourn at the seashore is highly to be recommended.

ACHONDROPLASIA[1]

(Chondrodystrophia Fœtalis; Fetal Rickets; Micromelia).

Congenital.

These terms are used to designate a peculiar type of congenital dwarfism arising from early fetal arrest of growth of the bones that are formed in cartilage, leaving the bones that are laid down in membrane unaffected. Thus, we have shortening of the extremities, and of the bones of the base of the skull, while the bones of the vault of the cranium and the trunk are normal. This peculiar chondral dystrophy produces the following characteristic statural disparities:—

Short extremities; long abdomen.
Trident hand.

Shortness of the extremities as compared with the normal (relatively long) abdomen; bowing of the extremities, especially lower, and thickening of the terminal epiphyses; limited power of extension of upper extremities; peculiar fan-like divergence of the thick, uniformly sized fingers, the so-called "trident hand"; marked narrowing of the pelvis; lordosis; protuberant abdomen; narrowing of the base of the skull ("pug-nose," broadening of the jaws), as compared with the normal (relatively large) upper part of the skull. The skin and nails are normal; the hair is soft and abundant in growth. Intellect is usually normal. The great majority of cases of achondroplasia die in utero or soon after birth. Those who survive may attain old age. They very rarely exceed four feet in height.

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[1] Though not an acquired disease, this subject is treated here in order to emphasize its many differences from rickets.

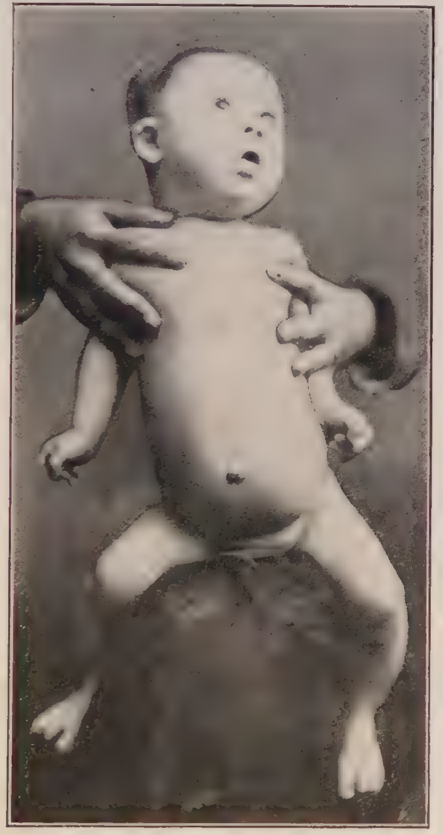

Fig. 161.—Achondroplasia (10 months old). Note length of trunk and shortness of extremities. (*Sheffield.*)

SCORBUTUS INFANTUM

(Moeller-Barlow's Disease, Acute Rickets).

Toxemia.

Malnutrition.

Infantile scurvy is an acute specific hemorrhagic affection of as yet unknown origin. It is probably due to direct microbic infection or toxemia resulting from intestinal putrefaction. As the disease occurs principally in infants from six to eighteen months old, the period when nutritional disturbances are most rampant, there is every reason to believe that malnutrition is the most active predisposing cause. This explains also the frequency with which infantile scurvy is observed in infants fed on boiled, sterilized or pasteurized milk (milk deprived of some of its nutritious qualities) or poor breast milk.

Usually sudden onset. The onset of the disease is usually sudden or, less frequently, preceded by malaise or digestive disturbance of a few days' duration. The child is restless, cries when it tries to move itself or when it is being handled. This symptom is the result of pain and tenderness especially in the lower extremities. For fear of pain the patient instinctively ceases to move its limbs (pseudo-paralysis). Examination of the extremities soon reveals at the diaphyses of one or both femurs, more rarely of the tibia and fibula, or upper limbs, spindle-shaped, colorless, smooth, non-fluctuating swellings surrounding the bones. The tumefactions

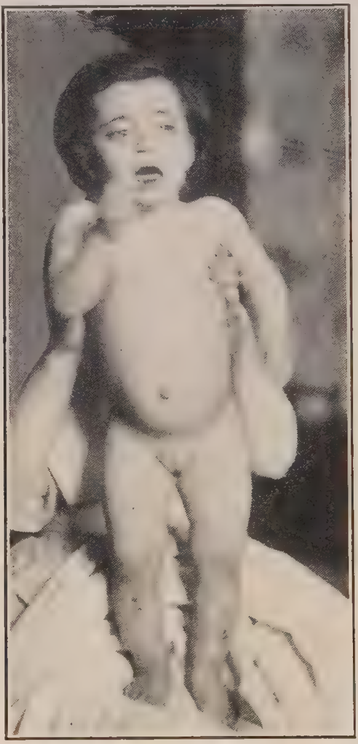

Fig. 162.—Moeller-Barlow's Disease (girl 15 months old). Note hemorrhage from the gums and in the skin and swelling of lower extremities. (*Sheffield.*)

Pain.

Pseudo-paralysis.

Tumefaction.

for the most part are due to subperiosteal hemorrhage. Exceptionally there is bleeding also from beneath the periosteum of the ribs and the bones of the head (protrusion of the eyeball, in subperiosteal hemorrhage of the frontal bone) and face, and occasionally spontaneous separation of the epiphysis from the shaft of the bone, leading to bone infraction, impaction or fracture. The next important symptom of infantile scurvy is sponginess and discoloration (minute transient ecchymoses) of the gums, with a tendency to bleed. In quite a number of cases the hemorrhagic tendency extends also to the skin, subcutaneous tissue (typical "black eye" after a fit of crying or laughing, also discoloration and proptosis of an eye resembling that of chloroma), mucous membranes and the viscera (dysentery!), so that as a result of loss of blood profound anemia, edema and albuminuria supervene. On the other hand, some cases pursue a very mild course (*formes frustes*), especially if recognized early and treated energetically. Except occasional permanent hyperostosis of the affected shafts the prognosis as a whole is favorable, recovery usually taking place within from a few weeks to as many months. Neglected cases, however, may end fatally from the aforementioned complications, or pneumonia.

Hemorrhages.

Spongy gums.

Antiscorbutic diet and fresh air form the treatment *par excellence*. Prompt improvement and rapid recovery usually follow the administration of fresh cows' milk, fresh fruit-juice (lemon, orange, or pineapple), beef-juice, and in older children fresh eggs and vegetables (potato purée, carrots, spinach, etc.). Where convalescence is protracted we may prescribe the syrup hypophosphites compound (U. S. P.), with extract of malt and cod-liver oil.

Fresh, nourishing food.

Fruit-juice.

Cod-liver oil.

Infantile scurvy may be mistaken for: Rheumatism, peliosis rheumatica, purpura hæmorrhagica, syphilitic epiphysitis, osteomyelitis, rickets and occasionally (when the orbit is involved) for chloroma.

Differential diagnosis.

In *rheumatism* the swelling is usually localized at the articulations and "jumps" from one place to another. It is accompanied by fever and responds to the salicylates. Hemorrhages are absent.

Peliosis rheumatica is characterized by deep red or bluish spots as a rule limited to the extremities.

Purpura hæmorrhagica is free from diaphyseal hematomas and pain.

Syphilitic epiphysitis is free from the hemorrhagic tendency, and often presents other syphilitic lesions.

Osteomyelitis is associated with high fever and local abscess.

Rickets is free from acute pain and hemorrhagic symptoms. Has other pathognomonic symptoms. It responds very slowly to treatment.

Chloroma or green tumor usually shows a predilection for the skull (temporal fossæ and orbits), giving the child a characteristic frog-like appearance. It is a grave blood disease—profound anemia with relative and absolute increase in lymphocytes.

DIABETES MELLITUS

(Glycosuria).

Frequent in children.

Within recent years, with increased interest in accurate diagnosis, the number of cases of diabetes in children recorded has greatly increased. In former years undoubtedly many of the rapidly fatal cases escaped observation. The importance of careful examination of the urine of older children and infants suffering from polyuria or enuresis, therefore, cannot too strongly be emphasized.

Dietetic diabetes.

We distinguish two forms of glycosuria: Glycosuria spuria (temporary or dietetic), and glycosuria vera (diabetes mellitus). The first variety is comparatively of little clinical importance. It is the result of consumption of sugar greater in quantity than can be assimilated, and usually disappears after arrest of the causal factor.

Diabetes mellitus.

On the other hand, *diabetes mellitus* is an extremely fatal affection, death taking place, in violent cases, sometimes after a few days, weeks or months, and in less acute cases often within a year or two at the latest.

Sudden onset.

Polyuria.

Glycosuria.

The onset of diabetes mellitus is sudden. The child begins rapidly to lose in weight, notwithstanding good appetite, suffers from excessive thirst, passes a large quantity of urine (often enuresis nocturna as well as diurna!), of high specific gravity (1030), containing a large proportion of sugar, and loses in vitality from day to day. In addition to these symptoms there are also digestive disturbances, skin affections (furunculosis, onychitis), cataract, nerve disorders (*e.g.*, often Friedreich's ataxia), obstinate acetone odor, dryness of the skin, etc. The course of the disease varies. As a rule, it is more rapid than in adults; the younger the patient the more violent the course. Death usually occurs as a result of general exhaustion or intercurrent diseases, such as pneumonia, tuberculosis, and the like, and is frequently preceded by coma diabeticum or uremia.

Coma.

Recoveries, however, are also on record. Every effort should be made to trace the cause of the disease and to combat it energetically. As congenital or acquired syphilis has frequently been found to play an essential part in the causation of diabetes, it is prudent to subject the patient to a course of antisyphilitic treatment. We have no means at our command to influence the other supposed etiologic factors of diabetes, such as traumatism to the head, shock, various infectious diseases, etc.; the time is not distant, however, when the true nature of the affection will be disclosed, and the remedies found which will greatly aid us in the prevention and arrest of the disease at its very inception. Until this blissful moment we will have to continue groping in the dark, empirically treat symptoms, and depend chiefly upon a restricted diet, which at best never strikes the root of the evil, and is hardly practicable in diabetes of early childhood. Wherever possible (especially in older children), the diet should consist of fresh meat-soups and broths; bread and biscuits of gluten flour, with cream and butter; eggs; moderate quantities of meats of all kinds, with spinach, asparagus, mushrooms, string beans, cabbage, radishes and turnips; fresh sour fruit, such as grapefruit, lemon, cranberries and raspberries. Saccharin instead of sugar. In infants milk and amylacea are indispensable, but should as much as possible be restricted. Oatmeal gruel seems to work well in some cases. Mild hydrotherapeutic procedures and light exercise are useful. Methylatropine bromid (gr. $\frac{1}{200}$) twice a day, hypodermatically; opium in some form, and arsenic, in addition to cod-liver oil and iron, are the only drugs of therapeutic value. Complications should be treated according to indications.

Symptomatic treatment.

Diet free from sugar and starch.

DIABETES INSIPIDUS

(Polyuria).

Polyuria, like glycosuria, may be transient or persistent. Transient polyuria is quite common in children and usually of nervous origin. On the other hand, persistent polyuria—diabetes insipidus—is comparatively rare. It is manifested by excessive thirst, polyuria (pale, sugar-free urine of low specific gravity), dry skin, disturbances of the digestive and nerve sys-

Transient or persistent.

Polyuria without sugar.

tems. The course is very protracted, but the prognosis *quoad vitam* favorable. Permanent recovery is rare.

Tonic treatment.

As the etiology is obscure (essentially the same as for diabetes mellitus), little can be expected from treatment, except in cases due to syphilis, which frequently yield to antisyphilitic medication. Change of air, hydrotherapy, and a nitrogenous diet act beneficially.

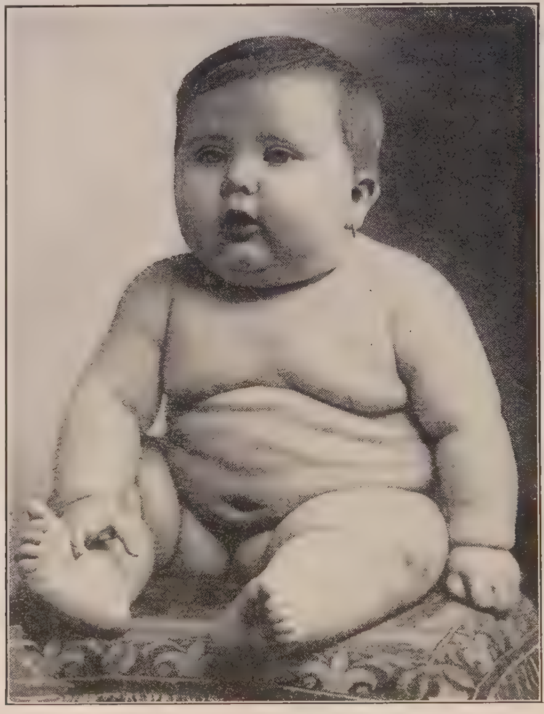

Fig. 163.—Adipositas (8 months old). Weighs 36 pounds. (*Sheffield.*)

ADIPOSITAS

(Lipomatosis Universalis. Obesity).

Spontaneous recovery the rule in infants.

Contrary to what is observed in older children or adults, overfatness in infants very rarely gives rise to constitutional disturbances. As a rule, the fatness subsides when the child begins to walk about.

In older children obesity is often associated with marked anemia, shortness of breath and fatty degeneration of the heart. If such symptoms appear, it is essential to eliminate fats and carbohydrates from the dietary and to recommend systematic exercise, active massage and hydropathic procedures. Carlsbad salts and thyroid gland substance are often useful; some cases, however, resist all sorts of treatment, and readily succumb to intercurrent diseases.

Diet.

Thyroid medication.

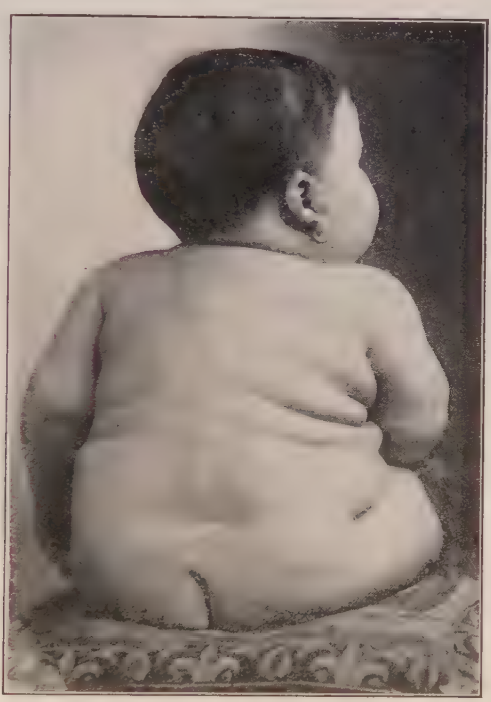

Fig. 164.—Adipositas. Same case as Fig. 163, back view. (*Sheffield.*)

Adipositas should not be mistaken for cretinism (*q. v.*).

CHAPTER XIV.

Diseases of the Nerve System.

GENERAL REMARKS ON CEREBRAL OR CENTRAL PARALYSIS AND BRAIN LOCALIZATION.

A symptom of brain disease.

"CEREBRAL PARALYSIS," so called, is not an independent brain disease, but merely a symptom occurring in connection with a number of congenital and acquired brain affections. Depending upon the extent of the lesion the paralysis may appear either in the form of hemiplegia, double hemiplegia, or monoplegia.

Unilateral lesion.

Spastic rigidity.

Involvement of cranial nerves.

Athetosis. Mental symptoms.

Hemiplegia is the result of a lesion (disease or trauma) in one cerebral hemisphere. The paralysis is situated on the side opposite that of the lesion. Motile power may be completely abolished or only partially so (paresis). Sensation may remain intact, but is lost if the brain lesion is in the internal capsule and extends to the sensory fibers. The paralysis is associated with spastic rigidity of the affected muscles; exaggeration of the deep reflexes; implication of some of the cranial nerves, such as the facial (paresis), hypoglossal (deviation of the tip of the tongue to healthy side), and ocular nerves (nystagmus, hemianopsia, and optic atrophy), and occasionally—in left-sided lesion—also with motor aphasia. As the paralysis becomes chronic the paretic musculature shows a tendency to arrest of development, tremor and athetosis; epilepsy and mental impairment up to total idiocy make their gradual appearance.

Bilateral lesion.

Crossed paralysis.

Double hemiplegia (diplegia) may be the result of two separate atacks of hemiplegia. More frequently it develops with one attack as a sequel of an extensive brain lesion in both cerebral hemispheres or in the pons and medulla (affecting both lateral halves). If only one side of the pons is involved we have crossed paralysis of extremities on one side and of the facial nerve on the other side.

In double hemiplegia, in addition to the symptoms enumerated under hemiplegia, functions may suffer which escape ordinary hemiplegia, *e.g.*, that of swallowing and, perhaps, that of micturition. Occasionally it is accompanied also by paralysis of the tongue, giving rise to symptoms which closely resemble those associated with bulbar paralysis. However, there is no wasting of the tongue, nor change in the electric reaction; hence, is spoken of as "pseudobulbar paralysis."

Paralysis of the tongue.

Monoplegia as a primary manifestation of a cerebral paralysis is rare. More frequently it is met in the regressive stage of the aforementioned two types of paralysis or in connection with lesions of the spinal cord or peripheral nerves. Cerebral monoplegia usually arises from a limited lesion in or near the cortex, less frequently from small capsular lesions involving individual nerve-bundles for the face, arm, leg, etc. (See also "Brain Localization," page 514.

In cerebral or spinal lesions.

The course of cerebral paralysis differs with the gravity and extent of the lesion. In cases of sudden onset which survive the immediate attack there is usually an early and appreciable improvement in the motor paralysis. The spasmodic rigidity may considerably improve or grow worse. The choreic and athetoid movements usually persist. The same is true of the mental impairment and of the posthemiplegic epilepsy, except that under suitable treatment there is some fair prospect to lengthen the intervals between the epileptic attack.

Improvement in motor paralysis.

Cerebral paralysis may sometimes be confounded with infantile spinal paralysis affecting one arm and one leg. The diagnosis can readily be cleared up, however, by bearing in mind the following differential points:—

Differential diagnosis.

Cerebral Paralysis.	Poliomyelitis.
Paralyzed limb rigid.	Flaccid.
Tendon reaction exaggerated.	Diminished or lost.
Electric reaction normal.	Diminished or lost.
Involvement of cranial nerves common.	Exceptional.
Atrophy of affected muscles slight.	Marked.
Athetosis common.	Absent.
Mentality affected.	As a rule not.

Monoplegia of cerebral origin differs from spinal in the same manner as hemiplegia. Besides, there is usually a history of preceding unilateral or bilateral paralysis with gradual improvement.

The treatment of cerebral paralysis is practically the same as in spinal paralysis: restoration of the sensory and motor power, and prevention of permanent deformities. Where the paralysis is due to local pressure (trauma, tumor, etc.), operative interference is indicated. A thorough course of antisyphilitic medica-

Anti-syphilitic treatment.

tion will not rarely be found a thankful experiment—regardless of discernible cause. Except in syphilitic cases, however, the prospects of a cure are very poor. The prognosis *quoad vitam* is fair, but depends upon the cause and treatment.

Brain Localization.

Seat of Lesion.	Usual Manifestations and Their Seat.
Central convolutions:	
1. Upper third.	Paralysis of leg, opposite side; convulsions.
2. Middle third.	Paralysis of arm, opposite side; convulsions.
3. Lower third.	
(a) Upper part.	Paralysis of the muscles of one-half of the face.
(b) Lower part.	Paralysis of the muscles of the lips and tongue.
Frontal convolutions.	Disturbance of speech.
Parietal convolutions.	Disturbance of cutaneous and muscular sensibility.
Occipital convolutions (especially cuneus).	Hemiopia; loss of visual memory.
Temporal convolutions.	Disturbance of hearing, opposite side, and sense of smell.
Centrum ovale.	Monoplegia, hemiplegia, hemiopia, word-deafness and aphasia; convulsions.
Central ganglia (caudate and lenticular nuclei).	Hemiplegia and hemianesthesia
Optic thalamus.	Disturbance of vision up to blindness.
Internal capsule.	Hemiplegia and hemianesthesia, and sometimes loss of special senses.
Corpora quadrigemina (anterior pair).	Oculomotor paralysis, reeling gait, possibly total blindness and deafness.
Crura cerebri.	Hemiplegia with crossed paralysis of oculomotor nerve.
Pons and medulla (one-half).	Hemiplegia with crossed paralysis of facial nerve; hemianesthesia; also involvement of other cranial nerves, *e.g.*, hypoglossal, abducens, varying with the height of the lesion.
Cerebellum.	Ataxia, vertigo, and vomiting.

PORENCEPHALIA.

Congenital or acquired.

Absence of brain substance may be congenital or acquired, occurring either as a result of embryonic arrest of development or of ante- or post-natal brain disease. The clinical symptoms arising therefrom depend upon the seat and extent of the defect, but generally correspond to those of pronounced microcephalus or hydrocephalus, *i.e.*, idiocy, hemiplegia, diplegia, defective speech, etc.

Idiocy; hemiplegia.

ANEMIA OF THE BRAIN
(Hydrocephaloid).

This condition is usually the result of excessive loss of body fluids (repeated hemorrhages), general grave anemia, exhaustion from acute (rarely chronic) gastrointestinal diseases, interference with the blood-supply of the brain (pressure on the part of tumors), etc. If the anemia is moderate, it is manifested principally by syncope.

Anemia of the brain occurring in violent gastroenteric affections (with profuse vomiting and diarrhea) is generally spoken of as "hydrocephaloid," so designated by Marshall Hall, who first described the symptom-complex. Hydrocephaloid is characterized by a stage of *excitation*: flushed face, fever, restlessness, jactitations; and one of *prostration*: pallor, sunken face, irregular pulse and respiration, cold extremities, subnormal temperature, sunken fontanelles, stupor with half-closed eyes, hazy corneæ, coma, convulsions, and, as a rule, death. Occasionally hydrocephaloid yields to energetic treatment, which consists of external heat stimulation by entero- and hypodermo-clysis, sterile camphorated oil and strychnin hypodermatically, champagne, and small quantities of food by mouth. Fresh air.

Stage of excitation; of prostration.

The brain of infants dying from cerebral anemia is pale, watery and softer than normal.

℞ Caffeinæ natrii benzoatis	gr. xij	0.8
Aq. destil.	ʒij	8.0

M. Sig.: Gtt. x, hypodermatically, for a child 1 to 2 years old.

HYPEREMIA OF THE BRAIN.

The hyperemia may be active, or arterial; or passive, or venous.

Active hyperemia may occur as a result of sunstroke, traumatism, mental or physical overexertion, overstimulation by exhilarating beverages or drugs, hysteria, onset of acute infectious diseases, etc.

Active.

It is manifested by deep redness of the face, congestion of the conjunctivæ, contraction of the pupils, hot skin, high temperature, accelerated pulse, strong pulsation of the carotids and temporals, ringing in the ears, intense headache, excessive thirst, and in severe cases convulsions, delirium, distention of the fontanelles, and other symptoms of meningeal irritation.

Congestion; fever, and convulsions.

Passive. *Passive* hyperemia of the brain is caused by passive congestion of the cerebral veins owing to cardiac debility, grave pulmonary affections (edema, pertussis, etc.), compression of the veins in the neck, etc.

Exhaustion. The symptoms of passive hyperemia are those of exhaustion, apathy, somnolence, cyanosis of the face and dyspnea.

Sedatives: stimulants. The treatment depends upon the original condition. It is more or less symptomatic—sedatives in active, stimulants in passive variety of hyperemia.

Upon the underlying cause also depends the final outcome. Protracted hyperemia sooner or later leads to meningitis, rupture of the blood-vessels, and dropsical effusion in the cranial cavities.

ACQUIRED HYDROCEPHALUS[1]

(Dropsy of the Brain).

By hydrocephalus is understood the accumulation of fluid within the cranium. The fluid may collect in the subdural space (external hydrocephalus) and be general or local ("sacculated"), or into the ventricles (internal hydrocephalus). External. Internal.

Clinically hydrocephalus may be divided into false and true. False. False hydrocephalus embraces all forms of dropsy of the brain accompanying active or passive inflammatory processes the intracranial pressure of which being insufficient to produce destruction of the contiguous brain tissues. It includes all cases of acquired hydrocephalus with a comparatively slight exudation, such as arise in connection with inflammation of the brain and meninges (tuberculous and non-tuberculous), acute infectious diseases with cerebral symptoms, severe gastrointestinal intoxication (acute and chronic), traumatism during or after birth, etc. True. True hydrocephalus is characterized by a primary deficiency (congenital!) or secondary (acquired!) destruction of brain tissue as a result of excessive pressure by a large exudation.

The symptomatology of *false* hydrocephalus resembles that of an acute or chronic inflammatory process of the meninges, or brain, or both, and depends not only upon the seat and amount of the effusion but also upon the course of the original affection. The principal symptoms are those of cerebral irritation, which may vary from simple irritability to marked convulsions, paresis, loss of vision, and coma. The symptom-complex is not a constant one, as is characteristic of true hydrocephalus. It may vary from day to day and may subside entirely with abatement of the original cause. The shape of the skull is but little changed. In infants the fontanelles are enlarged and bulging and the sutures are slightly separated. In older children with closed fontanelles no perceptible enlargement is discernible, except in progressive cases of long standing—in which event true hydrocephalus is then dealt with.

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[1] See "Congenital Hydrocephalus," page 124.

In false hydrocephalus symptoms inconstant.

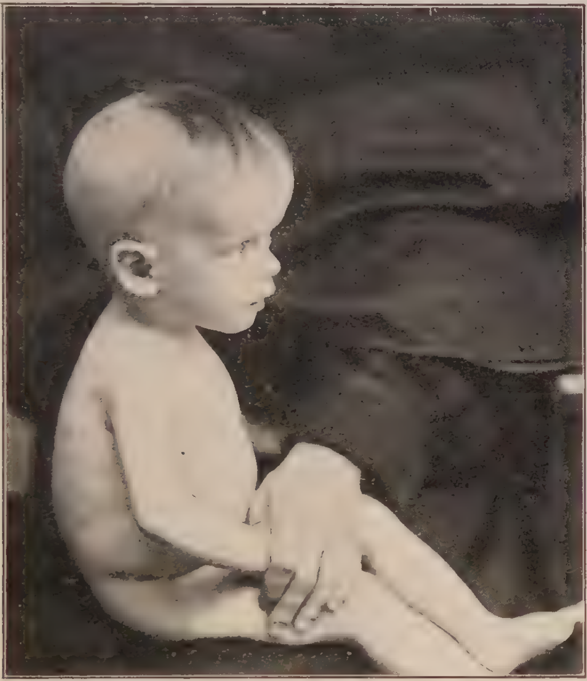

Fig. 165.—Acquired Hydrocephalus, following Acute Gastroenterocolitis. Patient also suffering from rachitis. See Fig. 156. (*Sheffield.*)

The course of *false* hydrocephalus differs with the etiologic factors. If the exudation is moderate and due to curable diseases, *e.g.*, gastroenteritis, traumatism, rickets, syphilis, etc., the further progress may be arrested and recovery occur. Some cases, of course, end fatally—with the underlying cause; others again, as previously mentioned, are transformed into true hydrocephalus, which is practically identical with "congenital hydrocephalus" (*q. v.*).

True hydrocephalus resembles the congenital variety.

INTRACRANIAL HEMORRHAGE

(Meningeal Hemorrhage, Hemorrhage in the Brain).

Seat of lesion.

We had occasion (see page 161) to direct attention to hemorrhages resulting from obstetrical injuries. This space will be devoted to the discussion of intracranial hemorrhages occurring during infancy and childhood. The usual sites for intracranial hemorrhages are as follows: Neighborhood of the large central ganglia, pons, meninges, convolutions, cerebellum, crura cerebri or medulla.

Trauma.

Syphilis.

Increased blood-pressure.

They may occur as a result of trauma, such as a blow or fall upon the head, in association with meningitis, infectious diseases, purpura, pertussis (as a result of severe venous congestion), sinus-thrombosis, syphilis (syphilitic arteritis), richly vascular tumors, nephritis and hypertrophy of the heart (owing to increased blood-pressure), etc.

Loss of consciousness; convulsions.

In the majority of instances the symptomatology is at first indefinite and inseparable from that of the fundamental disease. Where the hemorrhage is extensive, the symptom-complex resembles in its entirety that observed in intracranial hemorrhage in adults. Thus: Unconsciousness, convulsions; slow, irregular breathing; slow and full pulse, coma and death, or partial recovery with persistent focal signs, especially paralysis. (See "Cerebral Paralysis," page 512.)

The treatment consists of an icecap to the head, counter-irritation, perfect rest, light nutritious diet, and, later, ergot and the iodids. (See also "Central Paralysis," page 513.)

EMBOLISM OF THE BRAIN ARTERIES.

Valvular heart disease.

Cerebral embolism, like hemorrhage, is rarely observed in children. It is occasionally met in connection with severe valvular heart disease, and acute infectious and pyemic processes, and most frequently affects the arteria fossæ Sylvii.

Differentiation from cerebral hemorrhage.

The symptomatology of embolism is practically the same as in cerebral hemorrhage (*q. v.*), except that in the former the signs of cerebral compression and shock are not as persistent and as severe. Furthermore, the existence of valvular heart trouble decides in favor of embolism. The onset is usually sudden (occasionally preceded by headache, vomiting, etc.), with convulsions, coma, etc., followed either by early death or partial recovery, with remaining focal symptoms, especially hemiplegia and aphasia.

The treatment is the same as in cerebral hemorrhage. Antisyphilitic treatment may be tried in cases of doubtful origin.

SINUS-THROMBOSIS.

Thrombosis in the large sinuses of the dura mater is most frequently observed in debilitated infants. Two forms are distinguished: Passive or marantic, being the result of retardation of the venous blood-current in severe cardiac, gastrointestinal, or other exhausting diseases; active or infective, occurring in connection with inflammatory processes in the vicinity, *e.g.*, ear, nose, eyes, etc.

Debility.

Infection.

Passive sinus-thrombosis is usually limited to the longitudinal sinus and is manifested by symptoms of exhaustion and collapse and those of hydrocephaloid plus local edema and distention of the veins of the head and face.

Passive.

Active sinus-thrombosis usually involves the transverse and petrosal sinuses and is characterized, in addition to the aforementioned phenomena, by more or less marked septic symptoms (vomiting, chills and fever, etc.), hemorrhagic infarcts and embolism, *e.g.*, in the lungs, spleen and other organs of the body.

Active.

Differential diagnosis.

The differential diagnosis between the two varieties of sinus-thrombosis is quite difficult, but somewhat facilitated by lumbar puncture, which in the infective form reveals in the hemorrhagic cerebrospinal fluid numerous bacteria (strepto-, staphylo-, or pneumo-cocci). When the longitudinal sinus is involved we have: epistaxis, cyanosis of the face, frontal sweating; when the transverse and petrosal of one side: corresponding collapse of jugular vein and edema of mastoid region; when the cavernous sinus: exophthalmos.

Where a diagnosis can be established early opening of the sinus may prove a life-saving operation in septic sinus-thrombosis. Otherwise little can be accomplished in the way of therapy. In marantic sinus-thrombosis active stimulation may act well in some cases. The prognosis thus being so extremely grave, our attention should be directed principally toward prophylaxis, especially as regards extension of the suppurative process from neighboring structures.

Attention to suppurative processes.

POLIOENCEPHALITIS ACUTA (STRUEMPEL)

("Hemiplegia Spastica Infantilis," Bendix).

Resembles encephalitis and poliomyelitis.

The exact status of this diseased condition is still unsettled. Some authors look upon it as an irregular type of encephalitis, others group it with anterior poliomyelitis. As the clinical picture is so much at variance with either of these affections, it is, perhaps, preferable to treat it as a clinical entity. We should

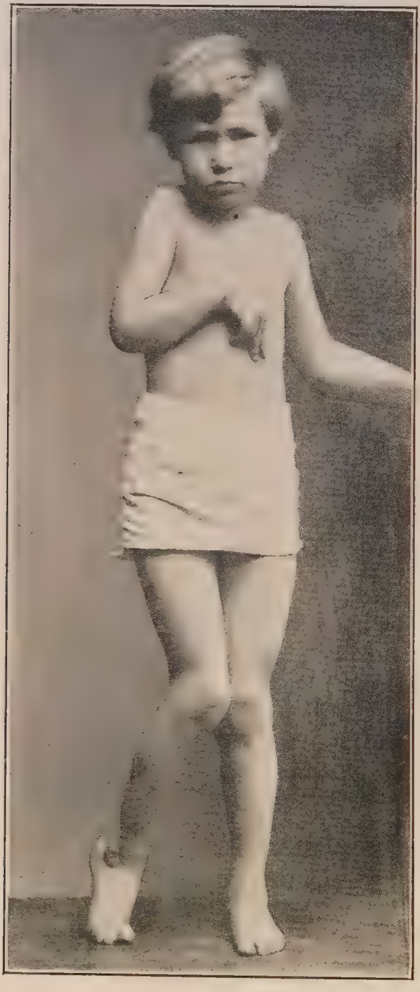

Fig. 166.—Polioencephalitis. Note peculiar position of right leg in the act of walking, and characteristic "athetotic" hand. (*Sheffield.*)

bear in mind, however, the fact that by extension of the inflammatory process either downward or upward encephalitis and poliomyelitis, respectively, may present a more or less identical symptom-complex.

Pathologic findings.

Pathologically, after abatement of the acute process, it is manifested by sclerosis, atrophy, fatty or cystic degeneration of certain portions of the brain—of several convolutions, an entire lobe, or of the large brain ganglia. These lesions are vestiges of inflammatory, embolic, thrombotic, or hemorrhagic processes within the gray motor cortical substance. Not rarely the pyramidal tracts down to the medulla spinalis exhibit secondary descending degeneration.

In early childhood.

It is a disease of early childhood, up to four years of age, and usually develops suddenly (very rarely insidiously) with fever, nausea, vomiting, headache and convulsions, or, less frequently in connection with other infectious diseases, such as exanthemata, pneumonia, etc. After subsidence of the acute course it is noticed that one-half of the body, or one arm or one leg is more or less paralyzed. Sometimes there are also disturbance of speech and mental impairment. As the disease progresses, the affected limbs become atrophied and contracted and manifest a great tendency to athetotic and choreic movements. The tendon reflexes are exaggerated, but the muscles never exhibit reaction of degeneration. Sensation is unimpaired. The cranial nerves (*e.g.,* facial), as a rule, are involved, but not to a great extent. In course of time especially under suitable treatment (which is practically the same as in anterior poliomyelitis) the paralysis, atrophy and contractures may somewhat improve and in mild cases disappear entirely, but on the whole the prognosis is bad. The patients are usually helpless in mind and body, are very prone to suffer from epilepsy and, where the cerebral symptoms are pronounced, rarely attain the age of 20 or 30 years.

Hemiplegia; contractures; athetosis and mental impairment.

Differential diagnosis.

As already suggested polioencephalitis may be mistaken for atypical encephalitis or anterior poliomyelitis. In both of the latter affections, especially in poliomyelitis, spasticity and athetosis of the extremities (both pathognomonic symptoms of polioencephalitis) are absent. Furthermore, in poliomyelitis there are reaction of degeneration and diminution or loss of tendon reflexes —the contrary being the case in polioencephalitis.

ENCEPHALITIS

(Inflammation of the Brain).

Non-suppurative (Hemorrhagic); Suppurative (Cerebral Abscess).

Trauma.

Extension of suppuration.

Encephalitis may be primary or secondary. Primary encephalitis usually arises as a result of traumatism to the head or infection by pathogenic micro-organisms, as is apt to occur in connection with divers acute infectious diseases, such as influenza, scarlatina, typhoid fever, measles, etc. Secondary encephalitis most frequently develops through extension of inflammatory or suppurative processes of neighboring structures, *e.g.*, the eyes (panophthalmitis), the nose (caries of the cribriform bone) and especially the ears (mastoiditis); as a result of pyemia, pulmonary abscess or gangrene, ulcerative endocarditis, embolism; foreign bodies in the brain, etc. The encephalitis may remain circumscribed or become diffuse; in either case, however, it may go on to suppuration.

Hemorrhagic lesions.

Encapsulated abscess.

In simple encephalitis the brain usually presents numerous minute hemorrhagic lesions. The larger foci at first appear red and soft, and later yellowish-white. After the process has run its course the affected part of the brain shows marked atrophy with cicatricial contraction. In abscess formation it is not uncommon to find, in recent cases, a cavity (one or several, small or large) filled with reddish or yellowish fluid, and in older cases encysted green, offensive pus. The abscess may remain encapsulated for a variable length of time (even years), and apparently do no harm, but may at any time perforate the sac, allowing the pus to permeate the brain substance, or enter the ventricles. The meninges rarely escape involvement.

Stupor, convulsions.

The clinical picture of encephalitis is very misleading and varies greatly with the seat and extent of the lesion and the stage of the disease. It is less confusing in cases of cranial traumatism, but, even in as severe an injury as fracture of the skull, the cerebral symptoms may be so vague as for days to escape notice. The onset is usually sudden with nausea, vomiting, fever, stupor and convulsions. Older children complain of dizziness and headache. This condition may last one or two days or as many weeks. Then either the coma increases and is followed by death or the symptoms abate, and the patient is apparently on the road to recovery, except that in the majority of

Paralysis.

instances monoplegia, or hemiplegia with or without involvement of some cranial nerves is left behind. The subsequent course of the disease depends upon the nature of the brain lesion. Simple encephalitis or suppurative encephalitis of very limited extent, with its cause removed, may clear up without appreciable after-effects. On the other hand, where an encapsulated abscess has formed, the violent symptoms may abate and the acute pass into a chronic stage. This state reached, the encephalitis is apt to run a very protracted course, with recurrent violent exacerbations and deceptive remissions, on the one hand giving rise to symptoms of acute meningitis; on the other, especially if the abscess is large and pressing upon the motor areas and cranial nerves, to those of tumor of the brain. In either case the diagnosis is often extremely difficult. Ordinarily *meningitis* differs from abscess in that it pursues a more acute course, and the brain symptoms are indicative of a more diffuse lesion. The diagnosis between *brain tumor* and abscess is still more difficult. In abscess there is usually an irregular temperature with rigors, motor aphasia and paraphasia, while in tumor fever is rare and there is a greater tendency toward disturbances in the area of distribution of the cranial nerves at the base of the brain, and toward choked disk. (See "Brain Tumor," page 524.) A history of ear disease or direct violence points strongly toward abscess. Slowly developing focal brain symptoms are characteristic of brain tumor. These differential points, however, at best, are not very reliable.

Differentiation from meningitis and brain tumor.

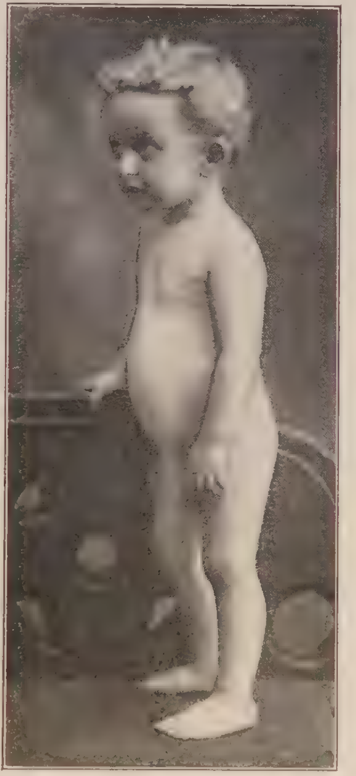

Fig. 167.—Encephalitis, with Left Hemiplegia. Note drooping of left shoulder and dragging of left leg in the act of walking. (*Sheffield.*)

As previously mentioned the remissions occurring during the course of chronic brain abscess are very deceptive. In the first place, the "latent period" is rarely entirely free from signs of ill health. As a rule, the patient suffers from occasional headache, vomiting, rise of temperature, mild paresis, etc. Secondly, there is no way of telling when in the midst of apparent good health the abscess may suddenly rupture in the brain ventricles or meninges and rapidly end fatally.

Latent course.

Sudden rupture of abscess.

The prognosis of brain abscess, therefore, is always very grave, unless surgical interference is resorted to early. The operative results are especially favorable in abscesses due to otitis or trauma—provided they can be localized.

Surgical treatment.

Hemorrhagic encephalitis, or purulent encephalitis before operation, should be treated by perfect rest, icebags to the head, lumbar puncture, etc.—the same as acute meningitis. Treated in this manner primary, simple encephalitis not rarely terminates in recovery.

Early prophylactic measures, particularly energetic treatment of ear trouble, scrupulous attention to suppurative conditions of the eyes, nose and throat are all powerful in the prevention of the dreadful complications and sequelæ.

Attention to ear disease.

TUMORS OF THE BRAIN.

Of the total number of cases of brain tumors on record about one-half occurred in children. Brain tubercle is especially common, and relatively frequent also are divers forms of sarcoma (gliosarcoma). Hidden as intracranial neoplasms are from sight and touch, their nature must necessarily be a matter of conjecture only, except, perhaps, in cases of bony growths, which may be diagnosed by means of the X-ray, and tubercle and syphilis, which may be surmised by the presence of other tuberculous or syphilitic lesions in other parts of the body or detected by the tuberculin or Wassermann's tests.

Tubercle.

Gliosarcoma.

Gumma.

The diagnosis of brain tumor is based upon the general and local nerve disturbances they produce. As a rule, the general symptoms precede the local, and consist of: Headache, vomiting, vertigo, optic neuritis, and convulsions.

Headache.

The headache is usually persistent, but may also be periodical, suggesting a malarial origin. The headache may be frontal, vertical or occipital, or equally distributed over all parts of the cranium. The locality of the pain occasionally bears a direct relation to the seat of the tumor, thus: when the growth is in the white substance the pain is usually frontal; when beneath the tentorium, occipital, etc. The same rule often applies to the pain elicited on tapping the skull over the seat of the disease. Intense headache in infants is indicated by rolling of the head from side to side, by throwing the hands up to the head, contraction of the eyebrows, and intolerance to light. The headache is frequently followed but may also be preceded by vomiting.

Vomiting.

The vomiting is projectile in character, and comes on suddenly. It differs from gastric vomiting by the absence of other signs of stomach trouble, and from vomiting accompanying migraine by the fact that the headache does not always terminate with it. Vomiting is especially characteristic of tumor in the medulla oblongata and in the middle lobe of the cerebellum, but it may occur in tumors affecting any part of the brain.

Vertigo.

The vertigo may be constant or paroxysmal and is most marked in affections of the pons or cerebellum. Vertigo in infants frequently escapes notice. It is manifested by sudden drooping of the head, pallor of the face and occasionally also vomiting.

Optic neuritis.

Optic neuritis sometimes forms one of the earliest symptoms of brain tumors. It does not always correspond to the size of the tumor. The neuritis is usually bilateral. It may develop slowly or rapidly, and in either case proceeds to complete optic atrophy.

Convulsions, general or local.

The child's nerve system being highly susceptible to irritation, increased intracranial pressure is quite early productive of convulsions of varying severity. The convulsions may be general or local. General convulsions with loss of consciousness may occur in tumors of any part of the brain, but are more common in tumors of the posterior fossa than in those of the anterior or middle fossa. Local convulsive seizures are met with chiefly when the neoplasm occupies certain situations. For example, convulsions beginning in the foot, as a rule, are indicative of the lesion being in the upper region of the motor area; those of the arm, the middle region, and those of the face, the lower region. It should be remembered, however, that the effects of a tumor may extend far beyond its actual site, and, furthermore, as the case proceeds, convulsions which from the outset have been local may become general. The convulsive attacks may recur frequently and last from several seconds to as many hours. The convulsions are not rarely followed by paresis or paralysis of the affected limbs. At first the muscular weakness may be transient, but as the disease advances it becomes permanent.

Paralysis.

The focal symptoms of brain tumors are manifested by uni- or bi-lateral hemiplegia, monoplegia, affections of speech, and paralysis of cranial nerves. The local symptoms pointing to the seat of a tumor attain their greatest precision when the swelling—be it a new growth or an inflammatory mass—is seated in the motor area of the cortex. They do not always correspond, however, to the size of the tumor. Furthermore, as the brain usually accommodates itself to the gradually increasing pressure and functional interference produced by the new growths, the appearance of the focal symptoms is frequently delayed until a very late stage of the disease. Once established, local symptoms are of great help in arriving at a correct diagnosis, except, perhaps, in cases where the tumor is multiple and distributed through various parts of the brain (*e.g.*, tuberculosis). See "Brain Localization," page 514.

Differentiation from tuberculous and syphilitic meningitis; brain abscess and epilepsy.

With the determination of the seat of the tumor, the diagnosis is greatly facilitated but rarely entirely settled. Brain tumors have several symptoms in common with tuberculous and syphilitic meningitis, brain abscess, epilepsy and hysteria; the differentiation between tuberculous and syphilitic tumors and chronic tuberculous and syphilitic meningitis is extremely difficult and often impossible, especially when the tumors are multiple. In *tubercle* and *gumma* the symptoms are more gradual in development, the optic atrophy more pronounced and the focal symptoms more marked and localized, while the course of tuberculous or syphilitic meningitis is more rapid. In *brain abscess* optic neuritis is less common, there is usually a history of ear disease, and after a period of "latency" it is usually accompanied by severe cerebral symptoms, fever and rigors (see "Encephalitis" page 522). *Jacksonian epilepsy* may resemble brain tumor in its early stage, but as the disease advances the diagnosis can readily be cleared up by the absence of optic neuritis and other focal symptoms. There are cases on record of *hysterical* hemiplegia with convulsions, and contractures which were mistaken for brain tumor. Careful investigation, however, will usually reveal the absence of optic neuritis, and the fact that in hysteria the symptoms are inconstant and multifarious, rather sudden in development and rarely progressive in character.

Detection of nature of tumor.

The nature of the tumor can sometimes be established by its seat. Thus, if the tumor is located in the cerebellum or pons, it is probably tubercle or glioma; if in the cortex, it is apt to be syphilitic. *Cysticerci* are most commonly met in the meninges or cortex. *Abscesses* are usually situated in the cerebral or cerebellar "hemispheres," and but rarely in the central ganglia, the pons, medulla, or the middle lobe of the cerebellum.

Anti-syphilitic treatment.

In view of the possibility of the tumor being syphilitic, it is always advisable to put the patient on an active antisyphilitic course of treatment (iodids and mercury, page 606). In syphilitic disease prompt treatment will soon be followed by amelioration of the symptoms, and, if faithfully persisted in, often by a cure. This therapeutic measure is occasionally attended by favorable results also in growths other than syphilitic, and should, therefore, be resorted to as a routine procedure in all obscure brain lesions.

Tonics.

Operation.

Should antisyphilitic treatment prove negative, and tonics in the form of fresh air, generous diet, cod-liver oil, iron and the hypophosphites fail to benefit the patient—tonics often do well in tubercle, and if employed early may in exceptional cases arrest its growth—the question of surgical interference should be taken under advisement. An operation is indicated where the tumor is single, and situated superficially in a part of the brain (motor area of the cortex) which can be reached and from which the tumor can be removed without immediate danger to life. Under favorable conditions, an operation should be performed early, before the general health has greatly suffered and permanent injury has resulted to organs and limbs from persistent brain pressure. Recently successful attempts have been made to remove growths from deeply seated structures; the results as to life and eventual cure, however, are still too few and too far between to warrant precipitate action.

In hopeless cases morphine and its derivatives will help to relieve agony.

SYRINGOMYELIA.

Congenital and acquired.

Paralysis.

Cavities in the cord may occur primarily as a congenital arrest of development or secondarily as a result of a gliomatous process in the gray (cervical enlargement) and white matter. In pronounced non-congenital cases it is manifested by gradual loss of power in the upper limbs, trophic disturbances in the skin, sub-

Trophic changes. cutaneous tissue, and bones (glossy skin, ulceration and necrosis of the phalanges), disturbance of sensibility (partial or complete loss of pain- and temperature-sense, while the muscular and tactile senses are preserved). Later, signs of muscular atrophy— Atrophy. beginning with a small muscle of the hand and gradually extending up to the shoulder—and paralysis, first of the upper then of the lower extremities, set in. The course of the disease is slow and occasionally interrupted by stationary periods.

SPINAL HEMORRHAGE.

The hemorrhage may be outside the dura, in the membranes, or Usually traumatic. in the substance of the cord. It is usually of traumatic origin—instrumental delivery, a fall or blow, severe convulsions. The history of the case, therefore, is valuable in the diagnosis. Slight hemorrhage may give rise to no definite symptoms. The diagnosis of severe hemorrhage is based on the sudden appearance of intense pain in the back, rigidity of the spine, sometimes convul- Pressure symptoms. sions and, if the pressure upon the cord is marked, paralytic symptoms (see "Myelitis"). The latter are especially pronounced Paralysis. in hemorrhage into the substance of the cord. Where the hemorrhage is moderate and the patient survives the immediate attack, the tendency of the affection is toward recovery. This may be enhanced by absolute rest on the face or side in a somewhat prone position. Local abstraction of blood, ice to the seat of the injury. Later, attention to the palsy.

SPINAL MENINGITIS.

In the majority of cases inflammation of the meninges of the spinal cord is associated with that of the brain (see "Cerebrospinal Meningitis," page 335). Occasionally, however, the inflammation is limited to the spinal membranes, like spinal hemorrhage, being produced by traumatism.

Pain, fever, rigidity and paralysis. The symptoms of spinal meningitis are practically the same as in spinal hemorrhage, except that the former affection is marked by a sharp rise in temperature at the onset, and by a more progressive character of the symptoms. Recovery is exceptional. The treatment is symptomatic.

SPINAL PARALYSIS

(Poliomyelitis Anterior, Infantile Paralysis).

As the name indicates the pathologic anatomy of this affection consists of multiple inflammatory foci (hyperemia, edema, infiltration of the small cells, swelling and cloudiness of the ganglion cells, destruction of the nerve elements, etc.), principally in the gray substance of the anterior horns of the spinal cord. Occasionally the inflammation extends to the anterolateral tracts and posterior horns, and while, as a rule, the lesion is limited to the cervical or lumbar enlargement or both, it may be found also in other regions of the cord and even in the medulla and pons—hence the diversity of the symptomatology.

Lesions principally in anterior horns.

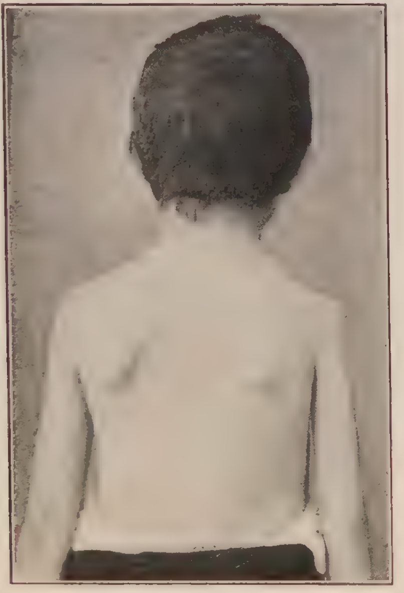

Fig. 168.—Anterior Poliomyelitis, Involving Right Arm. Note atrophy. (*Sheffield.*)

After abatement of the acute inflammatory process, some of the affected portions of the cord usually (there are but few exceptions) remain more or less permanently injured (atrophied), and it is upon the extent of this permanent—and not upon the initial—lesion that the further course of the disease depends.

Spontaneous recession of inflammation.

It is now generally agreed that the disease, whether it occurs sporadically or in epidemic form, is the result of invasion of the

Microbic origin.

Sudden, complete, flaccid paralysis.

Reflexes lost.

spinal cord by a micro-organism or its toxin. The onset is usually sudden. The local symptoms are preceded by systemic manifestations, such as rise of temperature, headache, muscular pain, drowsiness, sometimes convulsions and other grave cerebral symptoms. This initial stage may last from a few hours to several days and as the general symptoms disappear they are being replaced by the typical phenomenon of the disease—flaccid paralysis. The paralysis usually affects either both legs and one arm, one leg and one arm on opposite sides or very rarely on the same side, or both legs and both arms. Occasionally one extremity is affected, or only the muscles of the neck or abdomen. The paralysis is usually complete. The reflexes, both superficial and deep, are almost invariably lost. The faradic reaction is lost early, while the galvanic persists for some time. The paralyzed limbs are limp, flaccid, cool, and at times also cyanotic. The sphincters are almost always intact. In uncomplicated cases sensation is

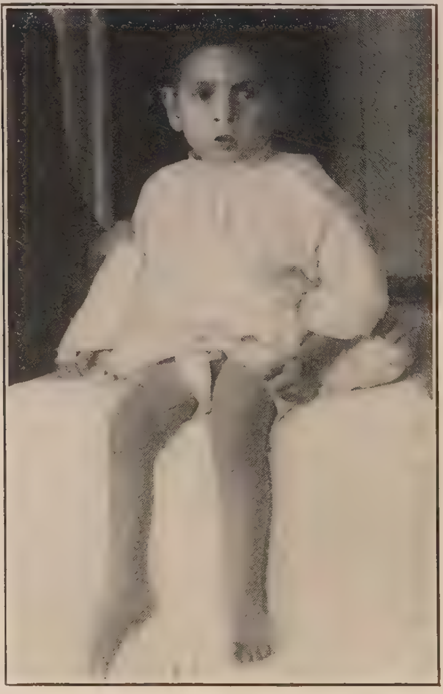

Fig. 169.—Poliomyelitis, Involving Right Leg. Note "foot-drop." (*Sheffield.*)

undisturbed, and there is no tendency to the formation of bedsores.) The paralysis does not remain long in its original intensity. Consonant with the abatement of the inflammatory process in the spinal cord, which usually occurs within a week, the paralysis begins to recede in one or more of the affected limbs, and at the end of a few weeks it is often limited to one or part of one extremity, to a group of muscles, or, in exceptional cases, to one or two muscles. If the paralysis does not disappear within the first few weeks or months, it usually persists for life. (The permanently paralyzed structures soon begin to waste and undergo fatty degeneration. The muscles are flabby and thin and the articular bands so lax that the limb appears elongated and is prone to slip out of joint. Frequently there is also atrophy of the bones.

Spontaneous recession of paralysis.

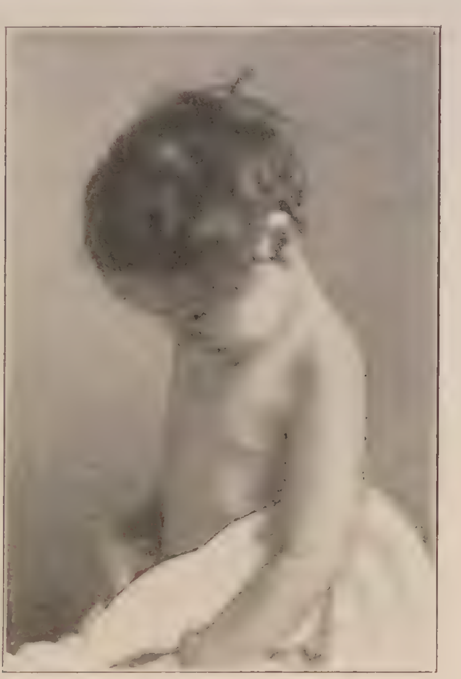

Fig. 170.—Poliomyelitis, Involving the Neck. Note forward "head-drop." (*Sheffield.*)

Atrophy.

As an immediate result of the atrophy of the diseased parts and the unopposed action (contraction) of the non-paralyzed antagonistic muscles, the affected extremities become contracted

Contractures.

Deformities.

and deformed—ordinarily for life, unless prevented and remedied by orthopedic and operative procedures. The deformities in the legs usually occur in the following order of frequency: Talipes equinus, equinovarus, equinovalgus, calcaneus or calcaneovalgus, and talipes varus. Manifold deformities arise also in the arms, neck and vertebral column from paralysis of the respective muscles (see Fig. 114). This is the typical course of the disease.

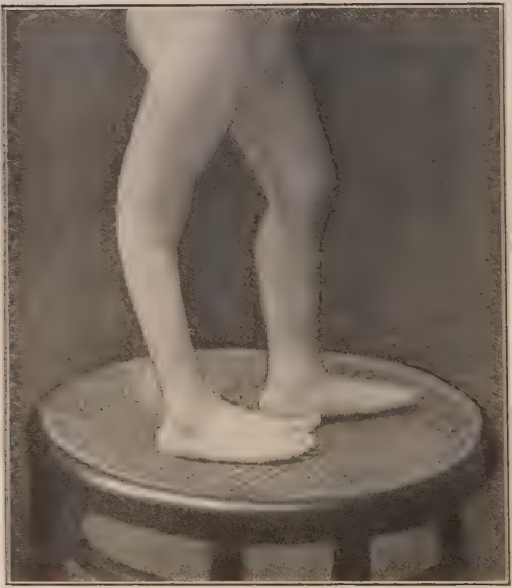

Fig. 171.—Anterior Poliomyelitis, Affecting Right Leg. Note atrophy and flaccidity of knee-joint. (*Sheffield.*)

Atypical cases.

Deviations from the typical course of the disease are not rare, and every epidemic is prone to present certain peculiarities. Thus, the onset may be either very mild or exceptionally severe. Where the onset is mild, the child may be found hopelessly maimed abruptly in the midst of perfect health. On the other hand, not rarely the initial stage is ushered in with vomiting, convulsions, stupor and similar meningeal symptoms, and continue for a week or so before revealing the exact nature of the affection. Furthermore the paralysis may develop in stages—at irregular intervals. In some cases paresthesia prevails; in others anesthesia—showing implication of the gray substance of the posterior horns. Occasionally the muscles of deglutition and respiration are affected, and where the lesion is situated in the medulla oblongata and pons the clinical picture of "polioencephalitis" (facial palsy, etc.) develops. Finally, some epidemics

Similarity to polioencephalitis.

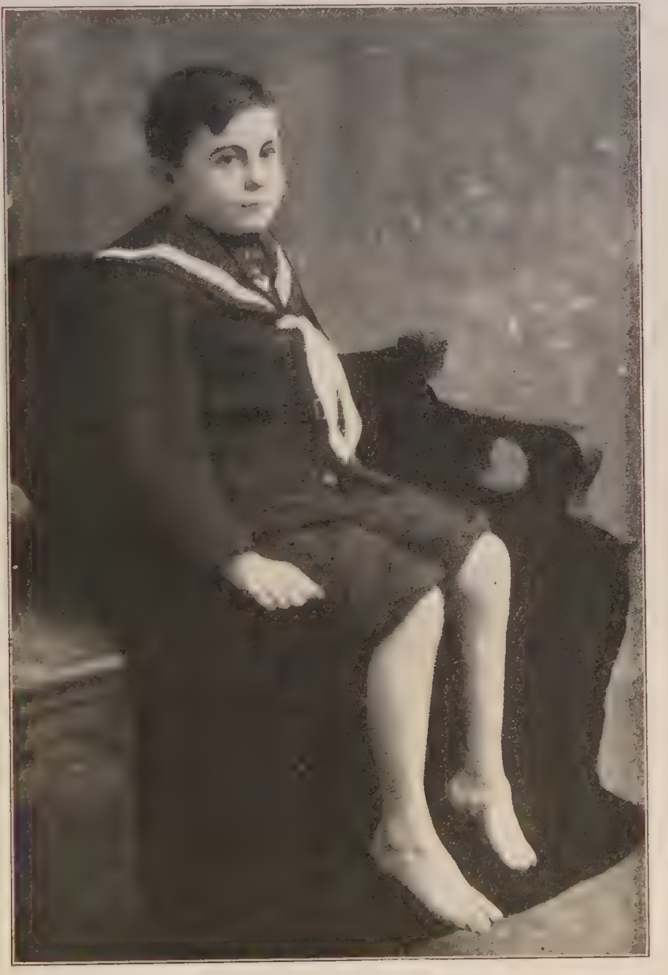

Fig. 172.—Paralytic Equinovarus in Poliomyelitis of Two Years' Standing. (*Sheffield.*)

are distinguished by prompt and complete recession of the apparently genuine paralysis.

Typical, fully developed spinal paralysis is strongly characteristic and presents no diagnostic difficulties. The initial febrile stage, the sudden appearance and spontaneous partial recession of

Pathognomonic symptoms

the paralysis, the almost constant integrity of the sphincters and the sensory sphere, the abolition of the reflexes and the electric (faradic) reaction and, finally, the appearance of muscular atrophy furnish a clear clinical picture. However, in the absence of an epidemic and where the case runs an atypical course, poliomyelitis, especially in its early stage, may be confounded with: Cerebral paralysis, polioencephalitis, myelitis, diphtheritic paral-

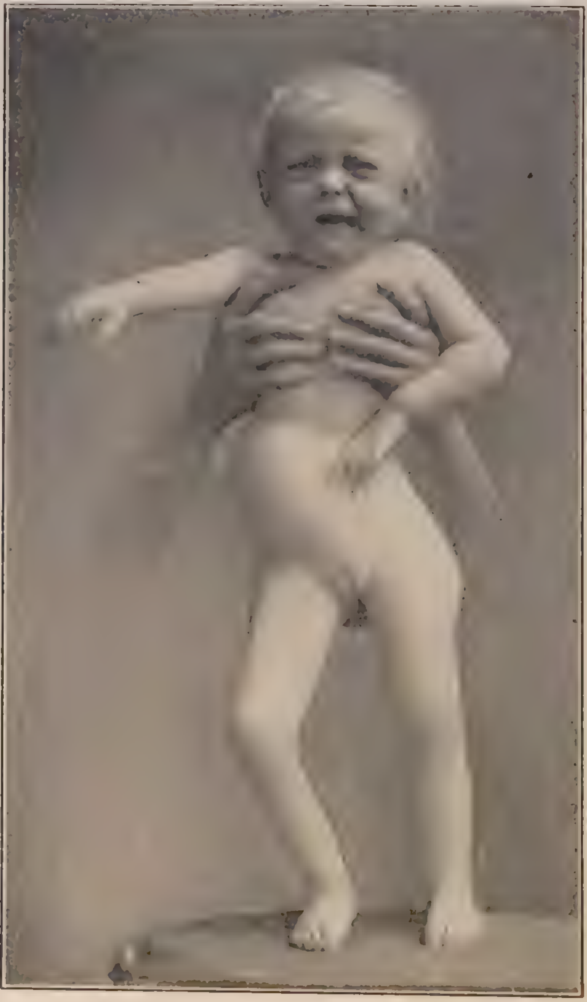

Fig. 173.—Anterior Poliomyelitis, Involving Extremities, Face and Abdominal Muscles. (*Sheffield.*)

ysis, and other affections associated with muscular and neural hyperesthesia and consecutive immobility of the affected limbs. The difference between cerebral and spinal paralysis has already been spoken of (see "Cerebral Paralysis," page 512). Severe poliomyelitis and mild *polioencephalitis* have many symptoms in common, and their differentiation is based principally upon the facts that in polioencephalitis the tendon reflexes are exaggerated and the muscles never exhibit the reaction of degeneration. Furthermore, spasticity and choreic and athetoid movements which are characteristic of the latter affection are absent in poliomyelitis. In *myelitis* the sphincters and the sensory sphere are almost invariably affected, and decubitus is quite common. *Diphtheritic paralysis* is preceded by diphtheria, is distributed symmetrically, and does not recede *en masse*, as is typical of poliomyelitis. During an epidemic, when our judgment is apt to yield to the anxiety not to miss the mark, *rheumatic* affections and *scurvy* may occasionally be mistaken for spinal paralysis. The presence of other rheumatic symptoms (tumefaction of the affected muscles or at the joints) in rheumatism and hemorrhages from the gums, etc., in scurvy, and, particularly, the absence of genuine paralysis in both diseased conditions are decisive.

Differentiation from polioencephalitis, myelitis, diphtheritic palsy, rheumatism, scurvy, Landry's paralysis and progressive muscular atrophy.

Chronic poliomyelitis may occasionally be confounded with *Landry's* paralysis (peculiar progress of the paralysis—no recession—normal electric reaction) and *progressive muscular dystrophy* (apparent hypertrophy in some muscles and atrophy in others, characteristic waddling gait, family proclivity to the disease. (See also page 545.)

faulty

With an early diagnosis we are frequently in position to limit the lesion to the primary focus and in part prevent all such deformities as arise from too early and strenuous use of the affected limbs. During the initial stage—*i.e.*, if the nature of the affection can at all be surmised—all such measures should be adopted as will insure perfect rest to the mind and body of the patient. The diet should be bland, the bowels kept open, the kidneys and skin active (principally by warm baths) and the patient preferably isolated, both to avoid transmission of the disease to others and to facilitate the enforcement of absolute restfulness of the patient. Medicinally, in addition to the warm baths, I place a great deal of reliance upon the abortive and curative value of the salicylates. It should be given in moderately

Rest in bed.

Warm baths.

Salicylates.

Massage.

Orthopedic appliances.

large doses all through the initial stage, and be followed by small doses of sodium iodid for a period of about six weeks. Medicinal nerve-tonics are in order later. As soon as the febrile symptoms have disappeared it is advisable to institute a course of local treatment consisting of gently stimulating baths, gentle massage, *passive* motion—to bring the paralyzed muscles into action—and the galvanic current (two or three times a week with the negative pole on the spine and the positive over the affected structures). This treatment should be continued for months. To prevent severe deformities of the lower extremities it is best to keep the patient off his feet for several months—until the paralyzed muscles have at least in part recovered their strength through the aforementioned mode of treatment. Above all, the child should not be allowed to run about without some sort of orthopedic apparatus to counteract the contraction of the antagonistic muscles. Old deformities demand surgical interference (tenotomy and tendon-transplantation) followed by the roborant mode of treatment just outlined. Persistence in the treatment is the keynote to success.

℞ Natrii salicyl.	ʒiss	6
Strychninæ sulph.	gr. ¼	0.016
Elixir simplicis	℥j	30
Aq. destil. ....q. s. ad	f℥ij	60

M. Sig.: ʒj every three hours for a child 4 years old.

MYELITIS.

Pathologic findings.

Cervical.

Dorsal.

This affection is occasionally observed in children principally as a result of traumatism, syphilis and compression of the cord by tuberculous masses and exudates between the dura and vertebræ secondarily to spondylitis. The pathologic process in the cord varies with the etiologic factors. Ordinarily the diseased portion at first is red and soft, and later yellow, fatty degenerated, atrophied and sclerosed. The lesion may be situated in any part of the cord and accordingly the symptoms differ with the localization. Thus, in disease of the *cervical region* there is first involvement (motor paralysis and sensory disturbances) of the upper extremities, then of the lower, and, if the lesion is very high up, the diaphragm also is affected and respiration is interfered with. In disease of the *dorsal portion* there is paraplegia (with muscular rigidity), with exaggeration of the reflexes, anesthesia of the extremities, paralysis of the bladder and rectum and decubitus. In myelitis of the *lumbosacral* region the paralysis, etc., is the same as in the former lesion; but the muscles are at first flaccid, then show degenerative changes to electric tests, then waste, and the skin and tendon reflexes are alike abolished. The feet fall into an extended position, so that the instep is on a line with the tibia. In *partial* myelitis the symptoms are less pronounced, extending only to such structures as are innervated by the diseased segment of the cord. In unilateral lesions the symptoms, of course, are limited to the side affected.

Lumbo-sacral.

The onset may be sudden or slow, according to cause. Acute cases set in with chills, moderate fever, nausea, sometimes vomiting and convulsions, radiating pain in the back and legs, rapidly followed by the aforementioned typical signs. Cases with gradual onset, *e.g.*, secondarily to spondylitis or compression by extraspinal growths, are manifested by gradually progressing debility of the muscles supplied by the spinal nerves below the compressed area, neuralgic pain, and disturbance of the bladder.

If the primary affection (*e.g.*, syphilis) can be reached and remedied before destruction of the cord has advanced too far, the progress of the disease can readily be arrested. Otherwise the symptoms continue to grow worse and at best can only be improved by massage, passive motion and faradization, procedures which are generally employed in all forms of chronic paralysis. Attention should be paid to the bladder (catheterization) and bowels, and particularly to the skin, as the tendency to the development of bed-sores is very great.

Symptomatic treatment.

ATAXIA HEREDITARIA (FRIEDREICH); HEREDO-ATAXIE CEREBELLEUSE (MARIE).

This family affection which is traceable through several generations is of obscure origin. Syphilis in the parents is the most probable cause. The anatomical lesion—degeneration—is situated principally in the cord (the column of Goll, and partly also of Burdach and Clarke) and in some cases also in the cerebellum. The cord as a whole is very thin and small, *i.e.*, arrested in development.

Often parental syphilis.

The disease attacks the patient insidiously, between the sixth and fifteenth years of life, with symptoms of simple progressive inco-ordination of the lower limbs, trunk, and arms—irregular swaying resembling that of chorea. Gradually the tabetic-cere-

Progressive inco-ordination.

Cerebellar gait.

General paralysis.

Mental impairment.

bellar gait develops, so that the child is ultimately unable to walk or stand. As the disease progresses, speech becomes peculiar, slightly scanning, heavy and awkward, vision disturbed by nystagmus, and occasionally optic atrophy (Argyll-Robertson symptom is absent, while Romberg's is occasionally present), the face expressionless, the general musculature paralyzed, atrophied, the spinal column curved, the feet lumpy-looking with the toes turned up (*Friedreich's foot*), and, finally, intelligence impaired. Unprovoked and uncontrollable laughter is said to be characteristic of the disease. As a rule, sensation and the cutaneous reflexes remain undisturbed; the sphincters intact until very late, while the tendon reflexes are abolished. The course of the disease is very chronic. The patient is usually bedridden after a period of from five to ten years, but he may continue to live in this state another ten years.

DISSEMINATED SCLEROSIS

(Multiple Sclerosis).

The etiology of diffuse and disseminated sclerosis is not definitely known. It is either congenital, and traceable to alcoholism or syphilis in the parents, or it is met in young, apparently healthy and normally developed children some time after traumatism or an attack of an infectious disease.

Loss of memory; scanning speech; spastic paraplegia.

Its onset is usually insidious with disturbance of motion, loss of memory, and dullness of intellect, soon to be followed by defective speech (at first slow and later scanning), hearing, and vision (nystagmus, amaurosis, and strabismus), spastic paraplegia (weakness and rigidity first of the upper extremities, then of the lower; exaggerated tendon reaction and ankle clonus) and intention tremor. In the later stages of the disease the patient loses control of the bowels and bladder, suffers from difficult deglutition, and attacks of vertigo, loss of consciousness and convulsions, and finally enters into a state of mental and physical exhaustion, paralysis and idiocy. Death occurs after several years.

The symptoms just enumerated do not all prevail in every case. They differ with the location of the sclerosed patches. As a rule, the latter are found not only in the brain but in the medulla and spinal cord as well—chiefly in the white substance. The disease is very rarely influenced by treatment. Antisyphilitic medication, however, is worth trying.

CONGENITAL RIGIDITY OF THE LIMBS
(Little's Disease).

The nature of spastic spinal paralysis is still obscure. Degenerative changes have frequently been found in the pyramidal tracts or their correlative structures of the encephalon. But whether these are the results of early antenatal arrested development (porencephalia), intra-uterine disease, traumatism during labor (embolism or hemorrhage), or simple prematurity are questions awaiting correct solution. Some cases are certainly acquired.

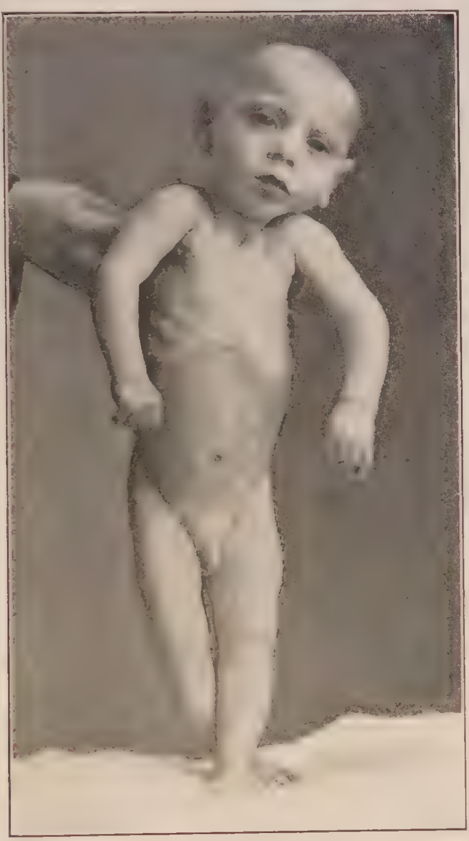

Fig. 174.—Little's Disease. "Scissors-gait" or cross-legged progression. (*Sheffield.*)

Lesions in pyramidal tracts.

The symptomatology of this affection is sometimes manifested soon after birth and sometimes not until the child begins to walk. Rigidity. One of the earliest symptoms is rigidity of the limbs. The child usually lies motionless (does not kick) with the legs pressed against each other or one upon the other. He begins to walk late and with difficulty or may not walk at all. If he is able to walk, Tiptoe-gait. he takes short rigid steps with the feet in tiptoe position, Scissors-gait. and the knees pressed closely together or crossing one another, sometimes half running so that at every step a fall seems imminent. The rigidity gradually grows worse, leads to fixed deformities and extends to the upper extremities and even the trunk. Z-shaped hand. A Z-shaped deformity is often observed in the hand when the patient attempts to use it. Early in the disease the deformities disappear during sound sleep or deep anesthesia. The knee-jerk is exaggerated, ankle clonus is generally present, atrophy is slight and develops late and the sphincters are normal. The majority of cases present symptoms of defective psychical development (up to idiocy), stammering nystagmus, strabismus, athetosis and epileptic convulsions. Where the latter symptoms prevail, the prognosis is very bad, otherwise it is not absolutely unfavorable. Symptomatic treatment. Under suitable treatment—stimulating baths, passive motion, massage and galvanization and later immobilization in the corrected position for a period of months, and, if this fails, tenotomy, tenectomy and tendon transplantation followed by the aforementioned therapeutic measures—the progress of the disease may be arrested and a partial cure obtained. Antisyphilitic medication is sometimes beneficial.

Differentiation from polioencephalitis. The differential diagnosis between this disease and polioencephalitis is based principally upon the absence (in Little's disease) of true paralysis and the presence of the characteristic, jerky, half-running, spastic scissors-gait.

TUMORS OF THE CORD AND MEMBRANES.

Tubercle. Neoplasms of the cord are very rare and, hence, principally of pathologic and diagnostic interest. They may be primary (sometimes congenital) or secondary. Tubercle is the most frequent Gumma. variety observed; next in frequency are gliomas, syphilomas, lipomas and sarcomas.

The symptomatology depends upon the seat of the growth, essentially resembling that of myelitis, except that it is of gradual development. In benign unilateral tumors the symptoms (motor and sensory paralysis) are limited to the side affected. Antisyphilitic treatment deserves full trial, and, if this fails, operative interference should be resorted to.

PERIPHERAL FACIAL PARALYSIS
(Bell's Palsy).

Facial paralysis may be due to trauma, pressure and irritation (swelling or disease) from contiguous structures, or exposure to cold or draughts.

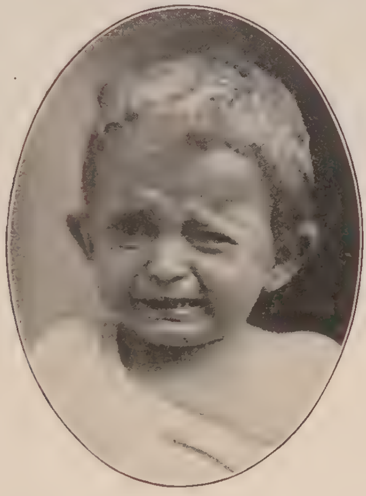

Fig. 175.—Peripheral Facial Paralysis—Bell's Palsy. Note inability to close right eye and drooping of right lower lip. (*Sheffield.*)

The symptomatology is essentially alike in all cases irrespective of cause. The paralysis is usually unilateral and affects the muscles of the forehead, the orbicularis oculi and some of the lower facial muscles. As a result of it the paralyzed side of the face is lax and expressionless, the nasolabial fold more or less effaced, the eye remains widely open and the angle of the mouth droops. The paralysis becomes especially pronounced, when the muscles are thrown into action, *e.g.*, on laughing or crying. In severe cases there is also paresis of the soft palate, and impairment of speech and mastication, and occasionally dullness of taste and diminished secretion of saliva. In otic facial palsy there may be disturbance of hearing (hyperacuteness). In the so-

Inability to close affected eye.

Distorted features.

Otic.

Rheumatic.

called rheumatic variety (due to exposure), the onset is usually sudden and accompanied by neuralgic pain. The electric reaction remains normal in mild cases, but is diminished or lost in grave cases.

Traumatic.

The prognosis and treatment depend upon the etiologic factors. Traumatic, especially obstetric facial palsy (*q. v.*), where the trauma is slight, usually ends favorably within a few weeks—without any therapeutic measures.

Dental caries.

Facial palsy arising from involvement of the facial nerve by aural suppurative processes (middle ear disease; caries of the petrous portion), usually runs a more protracted course, often long after removal of the cause. Early attention to the ear affection is of vital importance. Cases resulting from dental caries can readily be remedied by treatment, possibly extraction of the diseased tooth.

Pressure paralysis.

Rheumatic, grippal, etc., facial palsy ordinarily responds to local heat, the salicylates, quinine and arsenic. Pressure neuritis usually abates with disappearance of the tumor exerting the pressure upon the nerve. Facial palsy occurring in connection with parotitis calls for no special treatment. Where the pressure is due to a new growth, enucleation of the latter should promptly be undertaken. Recovery is not as rapid in the latter form as in the other varieties.

After abatement of the hyperacute symptoms a weak galvanic current should be applied four to six times a week, for from two to three minutes at a time. The anode should be held behind the ear, while the different facial nerve branches and muscles are stroked with the cathode.

Recovery assured with early return of electric reaction.

It has been observed that recovery is assured—after a shorter or longer period of time—in all cases of facial paralysis in which the electric reaction remains normal from the start or returns to normal after a lapse of from one to two weeks. On the other hand, cases which present complete reaction of degeneration of nerve and muscles after that period of time usually offer a doubtful prognosis. Protracted cases may lead to degeneration and shortening of the affected muscles, so that the face appears drawn to the paralyzed side.

Differentiation from cerebral and nuclear paralysis.

Peripheral facial paralysis should not be mistaken for central or nuclear facial palsy. In *cerebral* palsy the muscles of the forehead and eyes, for the most part, escape (*i.e.*, the patient is able to frown and to close the eye on the affected side); the electric reaction is retained; furthermore, the palsy is frequently associated with hemiplegia of the same side. In *nuclear* or basilar paralysis the palsy is usually limited to the lower half of the face (from the mouth down) and is complicated by other symptoms indicating a lesion in the pons, such as crossed paralysis and disturbed action of other cranial nerves.

POLYNEURITIS
(Multiple Neuritis).

Polyneuritis is an inflammatory, degenerative affection of the peripheral nerves. In severe cases the lesion ascends to the nerve trunks or even the roots. Its distribution is almost always bilateral and symmetrical. Polyneuritis is very rarely observed in children, since the principal causes of the affection—alcohol-, lead- and arsenic-poisoning—are of exceptional occurrence in young children. The most frequent form of polyneuritis encountered is that described as "Diphtheritic Paresis." (See "Diphtheria.")

Bilateral and symmetrical.

The onset of multiple neuritis is usually fairly rapid with numbness, pricking, pain and chilliness of the parts to be affected. This is followed by the appearance of motor inco-ordination (ataxia) up to paralysis of symmetrical groups of muscles (*e.g.*, of the hands and feet) or of entire extremities. The lower extremities are ordinarily affected first and the upper later. Genuine foot- and wrist-drop are rare exceptions. The same is true of involvement of the muscles of the trunk, and the sphincters. The motor symptoms are usually associated with sensory disturbances—pain, especially on pressure, along the nerve trunks, hyperesthesia and more rarely anesthesia. The electric and tendon reactions are diminished, and reaction of degeneration is quite common in severe cases. With early treatment—elimination of the poison (sodium iodid, magnesium sulphate, in lead poisoning), mitigation of pain (salicylates, warm baths), tonics (strychnine, iron, etc.), and galvanic electricity and massage—the prognosis is usually favorable, except when the respiratory muscles are affected. Occasionally atrophy, with consecutive contractures and deformities, may persist for a long time, and even for life.

Numbness, pain and motor inco-ordination.

Atrophy and contractures.

DIFFERENTIAL DIAGNOSIS.

	POLYNEURITIS.	POLIOMYELITIS	LANDRY'S DISEASE.
Onset	Usually slow. Slight fever, if any.	Quite acute; often vomiting. Moderate fever.	Slight prodromata (pain); no fever.
Distribution of paralysis	Symmetrical. Partial. Lower than upper extremities. Exceptionally other parts of body.	Irregular. Complete; often only one limb, or a group of muscles, *e. g.*, neck.	At first asymmetrical. Ascending. Complete. Legs, trunk, arms, and muscles innervated from the medulla.
Hyperesthesia	Persistent.	Transient.	Variable.
Anesthesia	Present (partial).	Absent.	Absent.
Atrophy and deformities	Late.	Early.	Very late, if at all.
Termination	As a rule, gradual recovery.	Partial, spontaneous, recovery.	Usually fatal within two weeks. Exceptionally, recovery.

The history of the case is very helpful in the diagnosis. Thus, in multiple neuritis, we are often able to elicit a history of some form of toxemia (infectious disease; lead-, arsenic-, or alcohol-poisoning); in poliomyelitis its prevalence in epidemic form may be decisive.

Differentiation from hereditary ataxia and myelitis.

Polyneuritis may occasionally be mistaken for *hereditary ataxia*—very slow in development, involvement of cranial nerves; mental debility; and *myelitis*—sphincters invariably involved.

HEMIATROPHIA FACIEI

(Progressive Facial Hemiatrophy).

The nature of this rare affection is still obscure. The pathologic findings point to an interstitial inflammatory process of the trigeminus. It occurs in girls more frequently than in boys, on the left side more than on the right, and exceptionally affects both sides of the face.

Begins at fossa canina. Atrophy of muscles and bones.

It begins with a small part of the face (usually over the fossa canina) turning white, thin, wrinkled, etc. From here the atrophy rapidly spreads to the muscles and bones of the entire half of the face, including the hair. At times the atrophy spreads to the chest and other parts of the body, but finally reaches a permanently quiescent stage. Sometimes there are also anomalies of pigment. It is occasionally associated with scleroderma and exophthalmic goiter.

Sensation remains intact and the electric reactions are normal. The cause of the atrophy being unknown, the treatment must, necessarily, be symptomatic. Paraffin injections have proved very useful to correct the remaining facial deformity.

HEREDITARY PROGRESSIVE MUSCULAR ATROPHIES

(1. Spinal. 2. Neural. 3. Myogenic).

This classification is intended solely to emphasize the principal locations of the underlying lesions. The disease is transmitted from generation to generation and often affects several members of the same family.

Family disease.

1. SPINAL PROGRESSIVE MUSCULAR ATROPHY.

It is observed in early infancy. It begins with weakness of the muscles of the legs, back, neck, throat, shoulders, arms, hands, fingers and toes. As the disease advances the muscles are completely atrophied (rarely pseudohypertrophied) so that the child is entirely helpless. The reflexes are abolished and the electric reactions greatly disturbed. The disease ends fatally within about four years from involvement of the respiratory muscles and consecutive pneumonia. The lesion consists of atrophy of the cells of the anterior cornu of the entire spinal cord and degeneration of the motor nerve fibers. There is no central involvement; hence, no cerebral symptoms. The sphincters are intact. Fibrillar twitching is infrequent.

Begins with the legs; spreads all over body.

2. NEURAL PROGRESSIVE MUSCULAR ATROPHY

(Peroneal Type).

It is characterized by atrophy beginning with the muscles of the legs, especially the peroneal group, and by predominance of sensory disturbances, hyperesthesia or anesthesia. In walking the child lifts the feet high and touches the floor with the tips. If the muscles of the hands are affected the hand becomes claw-shaped. Occasionally other muscles are implicated. The patellar- and Achilles'-tendon reflexes are at first diminished and later abolished. The electric reaction of the atrophied muscles varies—is normal in some cases, disturbed in others—irrespective of the state of the atrophy. Fibrillar twitchings are common. The course of the disease is very slow and interrupted by remissions of variable length, and judging by the underlying pathologic anatomy of the affection (degeneration of the respective peripheral nerves, with slight implication of the spinal cord) it is *per se* probably not fatal. Massage, baths and electricity are of benefit.

Principally peroneal group of muscles; occasionally hands.

Fibrillar twitchings.

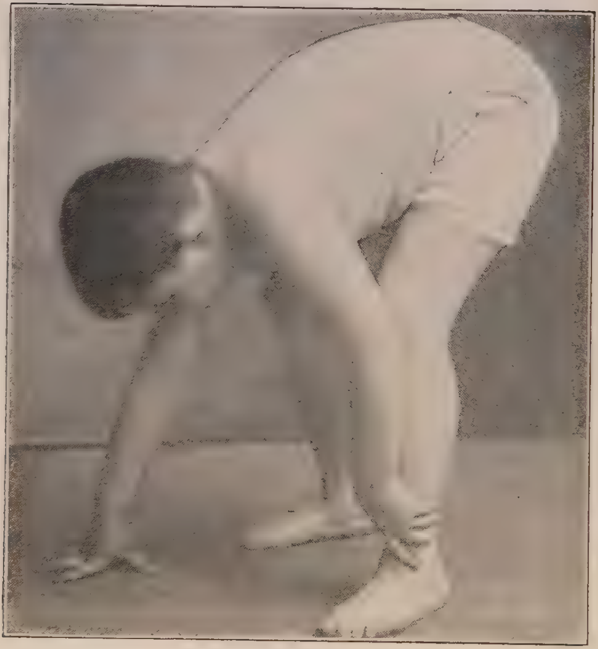

Fig. 176.

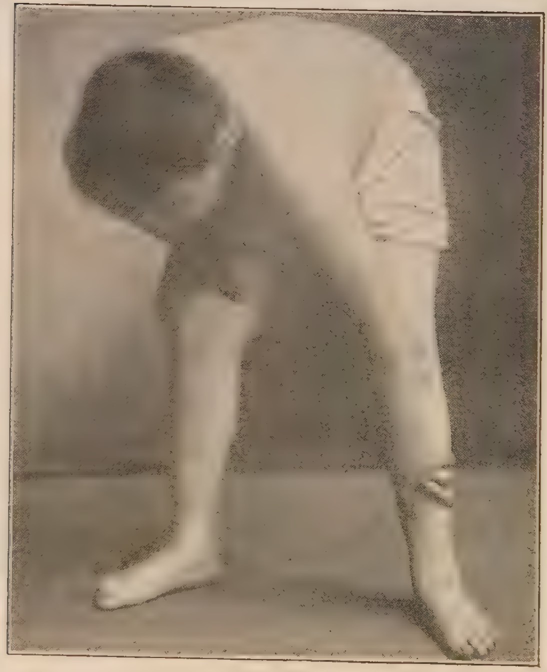

Fig. 177.

3. MYOGENIC PROGRESSIVE MUSCULAR ATROPHY
(Dystrophia Muscularis; Pseudohypertrophic Paralysis).

Under this heading are grouped the following four morbid conditions which were formerly looked upon as distinct pathologic entities:—

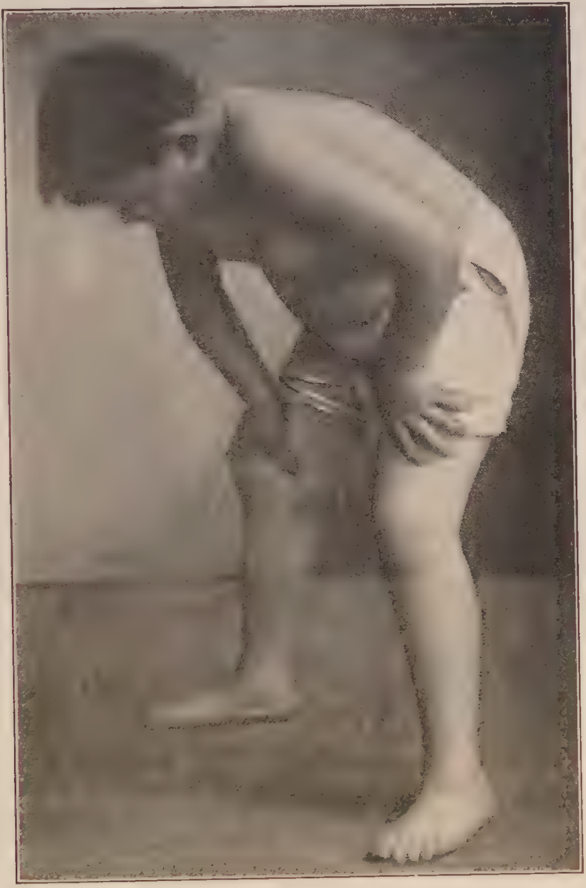

Fig. 178.

Figs. 176, 177 and 178.—Pseudohypertrophic Paralysis. Demonstration of mode of rising from the floor by "climbing upon himself." (*Sheffield.*)

(*a*) **Simple Hereditary Muscular Atrophy.**—It usually attacks children between eight and ten years of age, and is manifested by weakness and atrophy of the muscles of the back (without pseudohypertrophy), lordosis and paresis.

Weakness and atrophy of spinal muscles.

(*b*) **Infantile Muscular Atrophy — Facioscapulohumeral Type** (Landouzy-Dejerine).—As the name indicates it begins in early infancy with atrophy of the face, especially the orbicularis oculorum and oris and the lips. The patient is unable to close the eyes, to point the mouth, and his face becomes expressionless, like a mask. Pseudohypertrophy of the facial muscles sets in later, so also the atrophy of the muscles of the scapulohumeral regions.

In early infancy.

Affects face.

(*c*) **Juvenile Muscular Atrophy** (Erb).—The atrophy is manifested, at a later age than in the former variety, in the following order: The pectorals, the anterior serrati, the latissimus dorsi, the rhomboidei, and the trapezius muscles, and then the triceps, biceps, brachioradial and brachial muscles. The deltoid is usually strongly hypertrophied.

Muscles of chest and back.

(*d*) **Pseudohypertrophy** (Duchenne).—In this form of the disease the muscles first affected are those of the calves, the extensors of the thigh which become greatly enlarged, and then the long spinal muscles. As the disease progresses the shoulder, arm and lumbar muscles become involved, the deltoid, supra- and infra-spinati showing an especial tendency to pseudohypertrophy. The forearm and hands remain free. Owing to weakness of the erector spinæ and glutei muscles, the patient keeps his trunk thrown backward, "saddle-back," and walks with a peculiar waddling gait, with the legs widely separated and the toes barely touching the ground. The gait at times resembles that of bilateral dislocation of the hip. If placed on the floor, the efforts made to rise are very characteristic. Awkwardly and with difficulty he places first one hand and then the other on the legs, then on the thighs above the knees and in this manner he "climbs upon himself" until he assumes the erect position (see Figs. 176, 177, 178). In time, the patient becomes unable even to sit up.

Begins with calves of legs; later spinal muscles.

"Saddle-back": waddling gait.

Climbs upon himself.

The distinction between the different forms of myogenic dystrophia cannot always be made with exactness, as the order with which the atrophy begins is not rarely reversed. All varieties of the affection at a late stage present diminution of the tendon and electric reactions—but no reaction of degeneration or central disturbance. Fibrillary twitching of the atrophied muscles is absent and local vasomotor disturbances are rare. As the disease advances and the paralyzed muscles contract, various deformities (spinal curvature, talipes, etc.) make their gradual appearance and render the patient totally helpless and bedridden.

Slow course, with remissions.

The course of the disease is slow, and occasionally interrupted by remissions of variable length, and temporary improvement. Death usually takes place within ten years from the onset of the affection, as a rule, from intercurrent diseases, especially pneumonia. Treatment, in the form of baths, massage, etc., may prove effective to check the progress of the manifestations, but it is doubtful that it ever leads to permanent recovery.

Lesions also in cord.

The disease is attributed to an extraordinary increase of connective and adipose tissues with corresponding atrophy and gradual disappearance of fibers of certain muscles. Slight lesions are not rarely found also in the cord. The etiology is obscure. The absence of fibrillar twitching and of atrophy of the hands and forearms serve as differential points from "Spinal Progressive Muscular Atrophy." (See page 545.)

MYOTONIA CONGENITA

(Thomsen's Disease).

Rigidity on moving certain muscles.

It is a rare, probably hereditary affection of the muscular system, characterized by sudden spasm and rigidity of individual or groups of muscles, especially when the patient begins a voluntary movement, *e.g.*, arising from a certain posture, clasping hands, etc. Similar tonic contractions occur from the effects of a blow upon a muscle; and the application of a strong (20 to 25 milliampères) galvanic current produces certain wave-like muscle contractions which move from the area of the cathode to that of the anode. Although often appearing already in early infancy the disease does not endanger life or health. Warm baths and massage may prove of benefit.

Wave-like muscle contractions.

EPILEPSIA

(Epilepsy; Fits).

Pathologic alterations in the brain.

Epilepsy is an obscure affection of the brain, in typical form characterized by attacks of loss of consciousness, local or general convulsions, and a great tendency toward psychical disturbance. The situation and exact nature of the brain lesion is still undetermined, but, judging from the pathologic alterations (atrophy, hypertrophy, abscess formation, sclerosis, porencephalia, retention of subcortical cells, changes in the blood, etc.) so frequently found post mortem, there is reason to believe that there is no one pathologic entity responsible for the morbid condition.

Traumatism. Toxemia. Hereditary encumbrances.

The causes of epilepsy are many and diverse. Congenital defects of the brain or skull; traumatism to the brain or skull (during birth or after); infectious diseases affecting the brain directly or indirectly; toxemias of all kinds, including grave gastrointestinal intoxication; repeated attacks of convulsions from reflex causes; neoplasms, including syphilitic and tuberculous; sudden psychic disturbances, such as sudden shock, etc., among many other as yet obscure causes, all contribute their share toward the development of epilepsy at some period of life. An hereditary disposition is traceable in a certain number of cases, and children of syphilitic, alcoholic, and neurotic parents are more prone to contract the affection than those free from such encumbrances.

No age is exempt from the disease, but it is most apt to develop in children of from two to fifteen years old.

Rudimentary forms.

The exact time of beginning of the disease cannot always be traced, since the symptoms may be so mild as to escape observation. The child may for a few moments "hang its head," turn pale, and the paroxysm would be over with—hardly any reason to suspect epilepsy. The little attack may not recur for weeks or months, so that the last one is long forgotten when the next one sets in. It is only after the attacks grow longer in duration, stronger, more frequent, are preceded by an aura and possibly followed by involuntary urination and defecation, and profound sleep, that the nature of the dreadful condition is fully realized.

Aura

Genuine epilepsy varies greatly in severity not only in different individuals but also at different times. In addition to the rudimentary forms later to be described, the paroxysms are generally classified into severe (*grand mal*), mild (*petit mal*), and cortical or *Jacksonian*. The attacks are frequently preceded by a warning (*aura*) of motor, sensory or vasomotor character. There may be slight twitchings of the limbs, eyes, or head; slight general tremor, a vague sensation in the stomach, a feeling of numbness or pricking in the extremities, hearing of noises, seeing of colors or sparks, smelling of peculiar odors, irritability, hallucinations, etc.

Grand mal.

In *grand mal* immediately following the aura, and also without it, the patient, who may appear to be in good health, suddenly loses consciousness and falls, and becomes fixed in a tonic spasm,

Tonic and clonic convulsions.

with face and limbs contorted and breathing suspended. His face is pale or cyanotic; his eyes are widely open and staring or rolled upward or sideward. The teeth are pressed firmly together, with the tongue often impacted between them. In a moment the fixed spasm gives way to clonic convulsions. The face, body and extremities twitch violently, and the head beats strongly backward. During this stage the face is congested and often bathed in perspiration. Foam frequently fills the mouth, and may be mixed with blood from the severely bitten tongue. As the contractions cease, the child sinks down exhausted, limp and lifeless—except for deep sighing respiration—into a state of profound sleep (postepileptic coma) of variable duration. With return of consciousness he has no knowledge of what occurred. The duration of the paroxysms varies between one and five minutes. It may occur once or several times a day, a week or month, or may not return for several months and even years. A certain periodicity, however, is demonstrable in a great many cases. The attacks may also occur at night, during sound sleep.

Petit mal.

Petit mal is usually manifested by sudden loss of consciousness of very short duration. The patient may turn pale, stare vacantly, twitch a little, drop what he is holding, and then recover himself. Often in the midst of play the child suddenly stands fixed, "as if bewitched," with staring, absent-minded expression; a few moments later he resumes his play as though nothing had happened, or sinks down feebly or runs toward some object or person to support himself. The transition (sometimes after years) of petit mal into grand mal is not rare, and should always be remembered in fixing the duration of epilepsy.

Jacksonian.

In another group of cases the convulsions begin in one particular muscle or group of muscles, and rapidly spreads to other parts of the body. Loss of consciousness may be absent or occur after the convulsions have become general. It is often followed by localized paresis. This *cortical* or *Jacksonian* form of epilepsy is based upon a definite local lesion in ~~the cortex.~~

Epilepsy is not always represented by so typical a clinical picture. Rudimentary forms are encountered, which may tax the skill of even the best observer in reaching a correct conclusion. Two forms deserve special mention: Epilepsia nutans, and epilepsia procursiva.

Epilepsia nutans ("Salaamkrampf") is manifested by sud-

Spasmodic forward movements.

den lightning-like spasmodic forward movements[1] (between 20 and 100) of the upper part of the body—a sort of reverential bow—and is associated with partial or complete loss of consciousness.

Sudden forced running.

Epilepsia procursiva is characterized by a sudden forced start of running, of variable duration, which may cease abruptly or end in an attack of convulsions. Consciousness is partially lost during this seizure.

Mental fits.

In children as in adults, instead of typical or atypical attacks of morbid physical phenomena, momentary states of mental disturbances may occur which may vary from simple confusion up to acute mania. These fits occasionally alternate with convulsive seizures. Less frequently than in adults are the so-called post-epileptic—frequently rather proepileptic psychical aberrations which are manifested by unconscious, automatic, more or less violent actions, lasting minutes, hours or days. Inexplicable disappearance of children from home is not rarely an epileptic manifestation.

Post-epileptic attacks.

Permanent effects.

Epilepsy sooner or later leads to permanent mental impairment. In the earlier stages this may consist only of weakness of memory, silliness, alteration in the behavior (the child may be cranky, quarrelsome, destructive, etc.), but as the disease becomes chronic the patient's mental dullness increases and may reach a state of total idiocy. Furthermore, with the growing mental hebetude there is also a corresponding development of coarse features with a downcast, dazed, and stolid expression—physical peculiarities which to the keen observer often betray some hidden central lesion. This observation often serves well in the differential diagnosis between epilepsy and reflex and hysteroid convulsive paroxysms. (See "Spasmophilia" and "Hysteria.")

Prognosis favorable under suitable treatment.

The termination of epilepsy is subject to great variations. With the recent gradual improvement in the methods of diagnosis and treatment, complete recovery from genuine epilepsy is far from being exceptional. This refers particularly to cases due to reflex causes (defective vision, adenoids, worms, phimosis, etc.), when early detected and remedied. To a great extent this is true also of cases resulting from traumatism or benign neoplasms, which are nowadays operated upon more or less successfully. The surgical results are especially gratifying in the Jacksonian form of epilepsy. Operative interference, however, should always be preceded by an antisyphilitic course of treatment, which not rarely acts admirably. Some cases of epilepsy, after resisting all sorts of "cures" for a number of years, get well as unexpectedly as they got sick. Others again persist for life, do what you may. This is the so-called idiopathic epilepsy for which from time immemorial the whole pharmacopeia, witchcraft, mental healing, Christian or unchristian Science, etc., have been used in vain. What can be accomplished, however, in such cases is the lessening of the severity and frequency of the attacks. All sources of irritation, however trifling, should be removed. The patient should be placed on a light, salt-free diet, under the best possible hygienic conditions, and in the most congenial and restful surroundings. Residence in the country, with plenty of outdoor air, moderate exercise ~~and~~ hydrotherapy are ideal adjuvants.

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[1] Similar forward movements are frequently observed in divers forms of idiocy.

Reduction of attacks.

Attention to fit.

Immediate attention should be paid also to the convulsive fit, not alone to prevent a fatal issue from cerebral hemorrhage, or possibly from apnea, but principally to avoid grave bodily injury which the patient is apt to sustain during a severe fit. When the attacks are of frequent occurrence the child should not be left alone, especially in a room with an open fire, or in the vicinity of ponds, rivers, railroad tracks, etc., lest he be suffocated, fall out of bed, set himself on fire, drown, etc. A handkerchief or cork should be placed between the upper and lower molars to prevent biting of the tongue. A severe convulsive seizure may be aborted or modified by a few whiffs of chloroform, or amyl nitrite.

Bromids.

Of all remedies thus far recommended the bromids are the only ones which have proved of actual benefit in all forms of epilepsy. We should begin with moderate doses that will control the paroxysms. The bromids may advantageously be combined with small doses of Fowler's solution of arsenic. The treatment should be continued, with brief intermissions, to avoid bromism, for years—long after cessation of the attacks.

℞ Natrii bromidi,		
Ammonii bromidi	āā ʒij	8
Strontii bromidi	ʒj	4
Liquor potassii arsenitis	ʒss	2
Mist. rhei et sodæ	℥ss	15
Syr. aurantii	q. s. ad f℥iij	90

M. Sig.: ʒj in water every six hours, and later only twice a day, for a child 6 years old.

In severe fits we may add small doses of codeine.

When the bromids are not well tolerated by the stomach they may temporarily be administered per rectum. Postepileptic outbreaks frequently yield to early administration of hypnotics, especially chloral.

FUNCTIONAL SPASMODIC AFFECTIONS.

SPASMOPHILIA.

Eclampsia Infantum; Tetanism; Tetany; Pseudotetanus; Spasmus Glottidis.

Inherited disposition to spasms.

The subject in question is of great clinical importance, and still shrouded in mystery. Spasmodic affections are generally attributed to a number of local bodily irritations which act reflexly upon the central nerve system. We know this to be true. We know also that the infantile brain is very vascular, very irritable, very impressionable, lacking in power of resistance and control. We are in the dark, however, as to why the very same etiologic factors are prone to produce mild or severe convulsions in one child and none at all in the other. This apparent discrepancy in action leads one to assume that some children are born with a marked tendency to spasmodic affections. This, probably hereditary, spasmodic tendency ("spasmophilia") is distinctly traceable in children of nervous, alcoholic, syphilitic or tuberculous parentage, and exerts its influence principally on the group of functional spasmodic affections presently to be described.

1. ECLAMPSIA INFANTUM

(Convulsions).

Toxemia.

Reflex irritations.

Non-epileptic convulsions are of common occurrence in children, especially in infants under one year of age. They may occur as a partial phenomenon of all sorts of acute systemic disturbances, *e.g.*, toxemia from infectious diseases; gastrointestinal intoxication; shock, and trauma; or in consequence of continued reflex irritations, such as phimosis, adenoids, intestinal worms, intense pain from various causes, earache, teething, calculi, and the like. The frequency of the convulsive seizures is within no definite limits—from one attack in several months up to as many as thirty or more attacks in a day. The convulsions are both tonic and clonic in character. In the beginning the body is more or less rigid, the head and neck are retracted, the eyeballs are turned upward or roll spasmodically in different directions.

The face is distorted and grows cyanotic as breathing becomes labored or temporarily ceases. These tonic spasms are soon replaced by clonic convulsions—irregular and rapid twitching of the extremities and face or of single groups of muscles—which may last from a few seconds to several minutes, may remit and return with greater violence. With complete cessation of the convulsions the patient usually falls asleep, to wake up apparently free from cerebral disturbance. During the attack consciousness is lost. Occasionally there is loss of sensation as well as involuntary urination and defecation, foaming from the mouth and biting of the tongue—a group of symptoms which are generally met in epilepsy. This, together with the fact that eclampsia is not rarely a precursor of genuine epilepsy should put the physician on his guard in venturing a positive view as to the nature and curability of the spasmodic affection.

Tonic and clonic spasms.

Loss of consciousness.

Tendency to epilepsy.

Epilepsy differs from eclampsia in that the fit is preceded by an aura, that it is of short duration but non-remittent, and that it is invariably followed by profound sleep—not the light sleep which follows eclampsia. We should bear in mind, however, that these differential signs are much less reliable in epilepsy of children than in adults.

Differential diagnosis from genuine epilepsy, uremia and acute localized cerebral disease.

Eclampsia infantum is to be carefully distinguished from uremic convulsions, and spasms accompanying brain disease. In *uremia* there is usually a history (scarlatina?) of suppression of urine. The latter reveals evidences of kidney disease. *Cerebral convulsions* are associated with projectile vomiting, possibly a history of trauma, tuberculosis, otic abscess, and the like. The convulsions of organic brain disease are apt to be more localized, and be followed by paralytic phenomena.

When called upon to treat a child in an attack of convulsions, the physician is rarely in position to make exact and scientific discriminations between the different forms of convulsions. It is essential to arrest the convulsions irrespective of cause or effect, since a prolonged attack may end fatally from exhaustion or suffocation. The spasms are best controlled by means of chloroform inhaled from a handkerchief, moistened with ½ to 1 teaspoonful of the anesthetic. The anesthetic may be continued at long intervals for hours or days without endangering the life of the patient. As the convulsions subside, we begin to make careful inquiry into their causation and to employ the therapeutic measures indicated in each individual case. Hyperpyrexia calls

Arrest of spasm.

Symptomatic treatment.

for hydrotherapy (cold sponge or tub bath); gastroenteric disorders, for emesis (apomorphine 1/16 grain hypodermatically, or ipecac by mouth), catharsis (2 grains of calomel in one dose) and enteroclysis; intestinal worms, for teniafuges (turpentine inhalation, and calomel and santonin by mouth); nervous disturbance, for hot baths with or without mustard, bromids and chloral per rectum or by mouth, and counterirritation in the form of a mustard plaster or mustard-water cloths applied to the spine from the nucha downward. Lumbar puncture is a sovereign remedy in all forms of cerebral irritation associated with increased intracranial or intraspinal pressure and with the usual precautions can safely be employed in convulsions failing to yield to milder procedures.

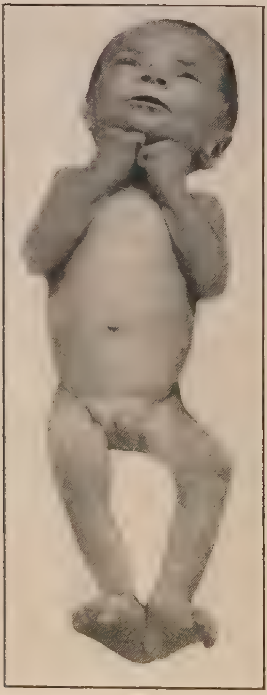

Fig. 179.—Tetanism. (See page 558.) During acme of spasm. (*Sheffield.*)

Prophylaxis.

With cessation of the convulsions due attention should also be paid to the more remote etiologic factors, principally with the view of prophylaxis. The diet should be regulated, the general health improved, rachitis promptly attended to, the faulty environment ameliorated, local irritations (*e.g.*, phimosis, adenoids, foreign bodies in ear or nose, rectal fissures, intense itching, etc.) promptly removed, and all such therapeutic measures instituted as will help to counteract and eradicate the inherent tendency to spasmodic affections.

Removal of local irritations

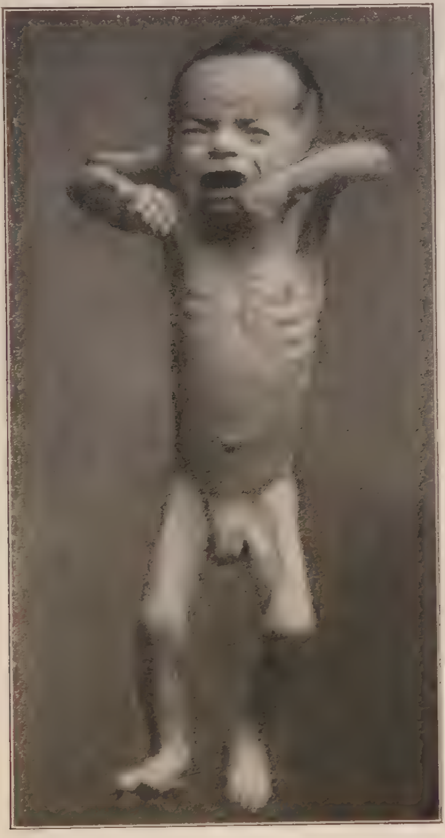

Fig. 180.—Tetanism. During partial relaxation of spasm. Same case as Fig. 179. (*Sheffield.*)

℞ Natrii bromidi	ʒj	4
Antipyrinæ	ʒss	2
Tr. ammonii valerianatis	ʒij	8
Syr. lactucarii	ʒiv	15
Aq. aurantii flor. q. s. ad	f℥ij	60

M. Sig.: ʒj every three to six hours for a child two years old. (General nerve sedative.)

2. TETANISM.[1]

More or less continuous.

This term is intended to denote a peculiar form of more or less *continuous* muscular hypertonicity occasionally observed in infants under three months of age. The affection is most probably due to gastrointestinal intoxication, since the infants suffering from it almost invariably are bottle fed, greatly reduced in vitality (often premature or syphilitic), subject to gastrointestinal derangement

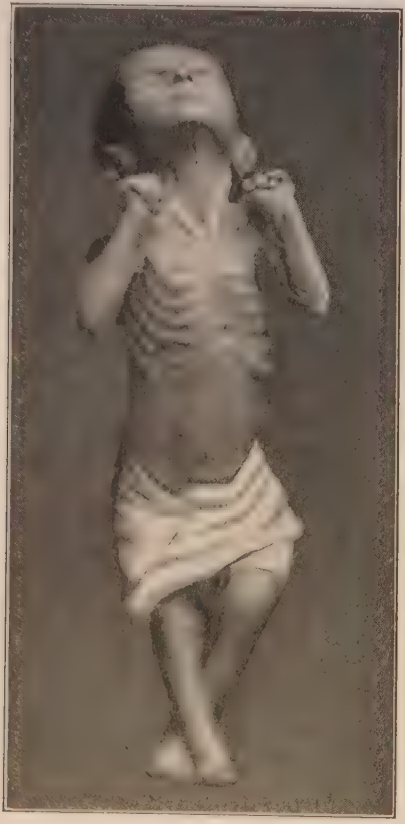

Fig. 181.—Tetanism. During acme of spasm. Note characteristic position of extremities. (*Sheffield.*)

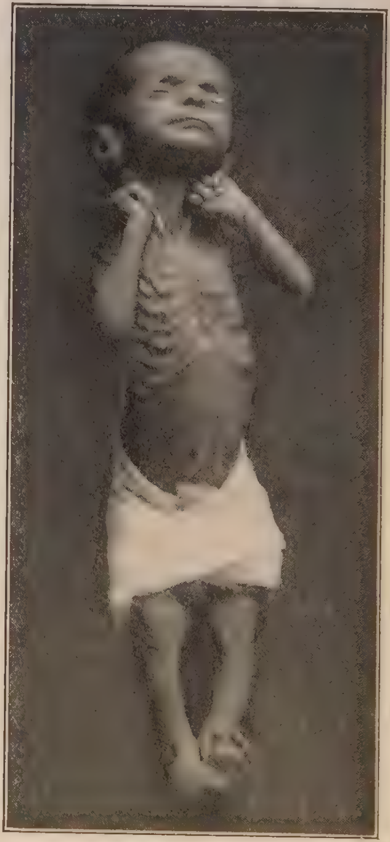

Fig. 182.—Tetanism. Same case as Fig. 181. During partial relaxation of spasm. (*Sheffield.*)

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[1] A similar or the same affection has been described by Hochsinger as "myotonia of the newly born and nursling." This designation is very misleading in view of its resemblance to "myotonia congenita" (Thomsen), which is an entirely different disease.

—in short present the clinical picture of profound marasmus. The onset of the spasmodic condition is fairly rapid. When fully established, the posture (see Figs. 179, 181) assumed by the infant is very pathognomonic. The head is retracted, the facial muscles are contracted, the jaws are firmly pressed together, the forearms are flexed upon the arms, while the hands are clinched so as to form closed fists. The rigidity of the lower extremities is less pronounced. As a rule, the legs are bent angularly, and the feet either overlap each other or are strongly arched. Now and then a partial relaxation of the spasm is observed (see Figs. 180, 182), and the spasm ceases entirely during sound sleep. The hypertonicity increases on handling the baby, but the "triad of tetany" is absent. The child is able to nurse without difficulty, in these respects differing from genuine tetanus and eclampsia.

Flexion of forearms upon arms.

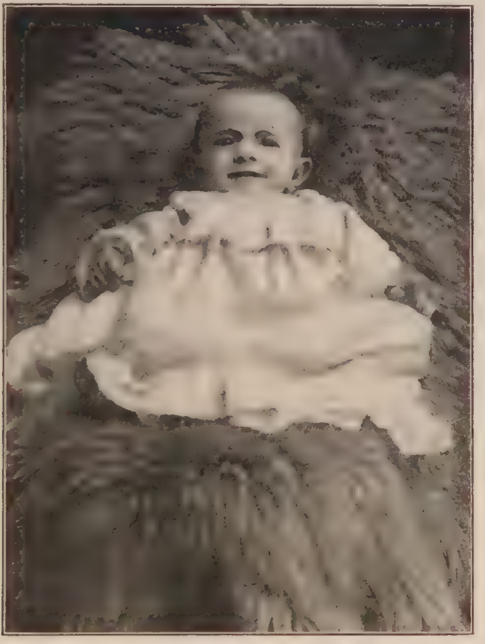

Fig. 183.—Same case as Fig. 181. Three months later. (*Sheffield.*)

Differentiation from tetany, tetanus and eclampsia.

With improvement in the general condition the spasticity gradually (within a week or a month or longer) subsides. Few babies survive, however, the persistent gastroenteritis and increasing exhaustion. The treatment is the same as in tetany, except that there is seldom an indication for the employment of hypnotics.

3. TETANY.

Intermittent contractures.

Bilateral and sudden.

Typical hand.

Latent form.

Trousseau's sign.

Chvostek's phenomenon.

Erb's sign.

This disease is characterized by intermittent somewhat painful contraction of certain groups of muscles, especially of the extremities, with exaggeration of the mechanical and electric irritability. The spasm is bilateral and usually sets in abruptly without loss of consciousness. The hands assume a very peculiar shape greatly resembling that of holding a pen or of an obstetrician dilating a tense cervix uteri (*main d'accoucheur*). Thus, the fingers are flexed upon the palms, the phalanges are extended, the thumbs are turned inward so as to be covered by the other fingers, and the wrists are flexed in pronation. When the lower extremities are affected the legs are adducted and the plantar surfaces of the feet are strongly arched, with a tendency to an equinovarus position. Occasionally the tetanic spasm extends to the neck and back, and exceptionally also to the laryngeal and other muscles of the body. On the other hand, cases of tetany are encountered in which the spasms are entirely wanting or barely indicated. These "latent" or passive forms of tetany may frequently be brought into activity by energetic *pressure* upon the main trunks of the nerves or vessels. This peculiar mechanical manifestation is spoken of as "Trousseau's phenomenon," and forms one of the three positive signs of tetany—the so-called "triad of tetany." The other two signs of tetany are those of Chvostek and Erb. "Chvostek's phenomenon" is based upon exaggeration of the mechanical irritability of the motor nerves, especially of the face (facialis phenomenon), and consists of lightning-like contractions of the face superinduced by *percussion* (with the finger or hammer) over a branch of the facial nerve while the face is in a state of perfect rest. "Erb's phenomenon" is based upon electric excitability of the motor nerves, so that a very slight *electric* current produces KaSz[1] or even KaSTe, if the current is but slightly increased. Sometimes AnOeTe and KaOeTe are obtained.

The duration of the tetanic attack varies from a few minutes to several hours or longer, and may recur once or several times daily or but once in several days. In the great majority of cases the disease usually subsides within a few days or a month or two, without any permanent sequelæ, provided suitable treatment is instituted early. The treatment, especially with the view of prophylaxis, is essentially the same as employed in rachitis—corresponding to the apparent relationship that exists between the pathogenesis of rickets and that of tetany. Like rickets, tetany occurs in infants chiefly of a half to two years of age. Like rickets, tetany shows a predilection for poorly fed and poorly housed children, and finally, as in rickets, the immediate cause of tetany seems to be some form of intoxication, intestinal or otherwise.

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[1] Ka stands for cathode; An, for anode; S, for closing; Oe, for opening; z, for weak contracture; Te, for tetanic contraction.

Related to rickets.

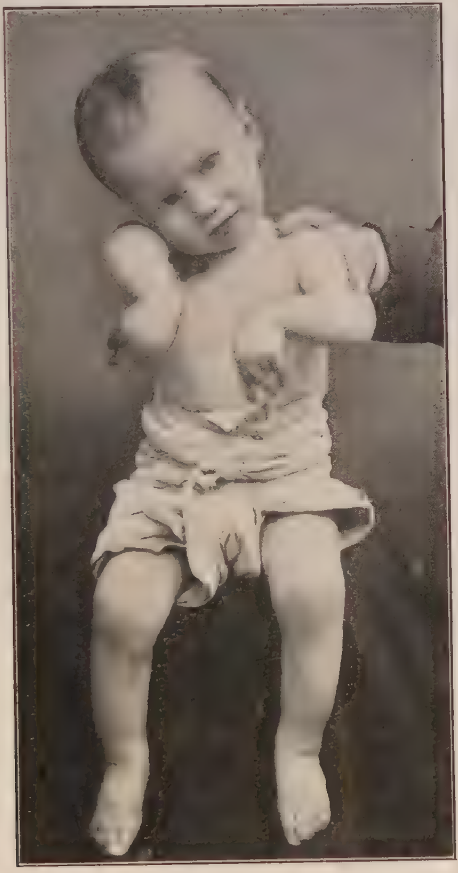

Fig. 184.—Tetany (child 11 months). Note characteristic attitude of hands. Slight contracture of feet. (*Sheffield.*)

Whether or not the immediate cause rests upon functional or organic disturbance of the thyroid gland or parathyroids is still subject to great differences of opinion.

Anti-rachitic treatment.

The diet should be regulated, as to quality and quantity. Young infants should, if possible, receive breast milk. The intestinal tract should be cleansed with calomel by mouth, lavage and high enemas. For the relief of severe contractions prolonged warm baths, bromids and chloral, will usually prove efficient. (See also "Rachitis," page 503.)

4. PSEUDOTETANUS (ESCHERICH).

Differentiation from tetanus, tetany and tetanism.

This affection differs from *tetanus* principally by its predilection for the muscles of the trunk, and by its afebrile course; from *tetany* by its spasticity being continuous, and from *tetanism* by the fact that it attacks children of from four to fourteen years of age (instead of infants) who are apparently enjoying perfect health. The pathogenesis of the disease is still unknown.

Arms and hands free.

The patients (usually boys) suddenly complain of stiffness in the legs and inability to walk about. The rigidity rapidly extends to the back and head, so that the patient lies motionless like a log, except for his ability to make free use of his arms and hands. The affected muscles are maximally contracted, prominent, and as hard as marble. The facial muscles except those of the eyes also are in a state of tonic spasm, so that the facial expression is that of trismus, the teeth are firmly set together and barely separable with force. Nevertheless, there is but little difficulty in feeding the patient. The rigidity is in partial abeyance during sleep as well as perfect rest, but greatly increased—up to painful opisthotonos, spasm of the diaphragm, etc.—by all sorts of bodily or mental irritations. During the height of the disease such spasmodic paroxysms may occur also spontaneously several times a day and are usually followed by profuse sweating.

Persists for weeks.

The spasmodic condition persists without apparent variation for from three to six weeks, whereupon the contractures gradually (within from two to four weeks) abate never to return.

The treatment is symptomatic (see "Tetany," page 558). Gavage, if necessary.

5. SPASMUS GLOTTIDIS
(Laryngospasmus).

Spasm of the glottis is a disease of infants of from six to twenty-four months old—the age in which rickets is most apt to prevail. It is closely related to and a frequent partial phenomenon of tetany, and seems also to rest upon the identical pathogenesis of the latter disorder.

Attacks of apnea.

The spasmodic attack is manifested by sudden deep inspiration, dyspnea, apnea, pallor and later cyanosis of the face, fixation or rolling of the eyes, and more or less marked rigidity of the body. At the end of a few seconds breathing is resumed after a noisy expiration. In severe cases the spasm not rarely extends to the diaphragm and to the entire musculature of the body.

Noisy expiration.

The attacks usually recur at shorter or longer intervals (several times a day!) and, if not terminating fatally—which may occasionally take place very suddenly even during a simple attack as a result of asphyxia—gradually subside after a few weeks or months. In mild cases recovery is the rule. The physician should be guarded, however, in the prognosis.

Differentiation from laryngeal stenosis.

Spasmus glottidis can readily be distinguished from other forms of laryngeal stenosis by its intermittency and noiselessness. It should not be confounded with the momentary apnea ("holding the breath"), frequently observed in children during a fit of crying. (See also "Congenital Stridor," and "Thymus Hypertrophy.")

Anti-rachitic treatment.

As the physician rarely has the opportunity to witness an attack of laryngospasm, his efforts must be directed chiefly towards its prevention. This is best accomplished by antirachitic treatment (*q. v.*), careful atention to the alimentary tract, and calming of the nerve irritability by means of small doses of sodium bromid (see "Eclampsia," page 555). Severe attacks call for stronger hypnotics.

A severe attack may be aborted by dashing cold water in the child's face, exciting choking motions by pressure upon the root of the tongue, and exciting sneezing by irritating the nasal mucous membrane. Timely intubation and artificial respiration have saved some babies from immediate death.

CHOREA VERA
(St. Vitus's Dance).

Microbic origin.

Genuine chorea is an acute, infectious, sporadic and epidemic affection characterized by spontaneous, irregular movements of the voluntary musculature, and by a special tendency toward cardiac complications.

Related to rheumatism.

The specific causal micro-organism of this disease is still unknown, but is probably closely related to that of rheumatic affections, with which chorea is occasionally associated. Other infectious diseases (such as exanthemata), fright and mental overwork serve as predisposing causes.

Grotesque, involuntary muscular movements which cease during sleep.

Involvement of heart.

The onset of chorea is preceded by prodromata varying in duration from a few hours to a few days. They consist of fretfulness, fatigue, restless sleep and occasional twitching. After the prodromic stage the actual attack may be precipitated abruptly and with full force, or come on gradually and run a mild course. The cardinal symptoms of the disease are irregular, awkward, involuntary, muscular movements—hasty and beyond control—which cease only during sound sleep. The movements intermittently involve various sets of muscles, never letting up a moment while the patient is awake. The movements are intensified when the patient is conscious of being observed, and tries to control them, or attempts to perform some voluntary action. The shoulders, one or both, jerk upward or downward; the arms rotate from side to side, or are forcibly thrown backward or forward; the hands are engaged in incomplete extension, flexion, pronation or supination, while the fingers are bent, extended or shoved one over the other so that the patient is unable to hold an object firmly, to write, to button a garment, etc. The head sways from side to side, often describing a semicircle, or is dropped downward so that the chin touches the chest wall. The facial muscles twitch, and produce grotesque distortions of the face and mouth. The forehead is wrinkled, the eyes open and close, the patient seeming to cry or laugh. In one case under our observation the iris (!) was involved so that the pupils contracted and dilated almost incessantly. The tongue participates in the movements, causing difficulty in eating and drinking, and defective speech up to aphasia. The movements of the lower extremities vary with the intensity of the attack, in severe cases being of such nature that the patient is unable to stand, sit or lie still, and frequently falls, stumbles, or is thrown out of bed and injured. During the acme of the attack it is not uncommon to find irregular respiration and arhythmia of the pulse—both from implication of the respiratory muscles and the heart (*chorea cordis*). Notwithstanding, however, the intensity of the movements the patients rarely complain of being fatigued, in fact a great many children are otherwise in perfect health. The temperature is normal, the digestion good, sensory disturbances are usually rare and slight (hyperesthesia along the course of the nerve trunks), the patellar reflex is somewhat exaggerated, but the cutaneous sensibility and reflexes are unaltered.

Self-limited.

If left untreated the active stage of the disease lasts from four to six weeks, then the symptoms gradually diminish and may disappear entirely a few weeks later. Some cases run a mild course from beginning to end, at no time presenting the aforementioned grotesque muscular excursions. This is especially prone to occur if treatment is begun early, and persisted in.

Complications.

The intensity of the atttack stands in no relation to its duration; on the contrary, cases of slow development and moderate severity may run a chronic course and suffer relapses, while violent cases often respond to a few weeks' treatment. This incongruity is often observed also as regards complications, mild cases being not rarely associated with inflammation of the joints, pleura, pericardium or endocardium, whereas severe chorea may run its course without any untoward result. In reference to heart complications it is well to remember that not every blowing heart sound heard in chorea is indicative of a valvular lesion—the majority of these adventitious sounds disappear, perhaps, never to return. On the other hand, heart lesions have been found at the autopsy without any indications of their presence during life, a fact which strongly emphasizes the necessity of prophylactic measures being taken against heart disease (perfect rest) during the active stage of the disease.

Hemichorea.

Paralysis.

Sometimes the muscular disturbance is limited to one-half of the body (*hemichorea*), showing that the lesion is localized in one hemisphere of the brain. This form of chorea is more serious than bilateral chorea. It is often associated with paresis of the extremities, one or both (*chorea paralytica; chorea mollis*), and changes in the psychical condition, *e.g.*, melancholy, hallucinations.

Recurrence.

Notwithstanding the grave nature of the affection the prognosis of chorea, on the whole, is favorable. A fatal termination is exceptional. It may occur either as a result of complicating heart disease, or from some, as yet unknown, effect upon the central nerve-system. To the latter class belong the cases associated with delirium and prostration. On the other hand, the prognosis as to permanent recovery is not quite promising. Recurrences are frequent, and as previously mentioned the tendency to permanent heart disease great.

Epidemicity.

Pseudo-chorea.

With these facts in view, the urgency of instituting preventive measures against chorea is obvious. This is strongly emphasized by the observation that chorea may appear in epidemic form (it is quite common to find several members of one family to be attacked simultaneously or within a brief period of time). I am not referring to the hysterical "pseudochorea" not rarely encountered in epidemic form in girl's boarding-schools (see "Hysteria," page 588). Prophylaxis is best accomplished by isolation of the patient. This is imperative in hospitals, asylums or private schools where several inmates are congregated in close quarters. Girls (between 6 and 12 years of age) particularly should be kept apart, as they are very susceptible to chorea—about 70 per cent. of the cases being met in girls.

Physical and mental rest.

Hydrotherapy.

Arsenic in large doses.

Tonics.

Salicylates with digitalis.

Hypnotics.

The active treatment consists principally of perfect rest in bed in an airy and sunny room, and avoidance of all mental excitement. While the choreic movements are very pronounced, the patient should be kept in a well-padded bed (to avoid injury) day and night, but, as the symptoms improve, she may be allowed to sit up or be around and about for a few hours at a time. A warm bath with a cool sponge once or twice a day and a daily colon flushing are very salubrious. The food should be bland, nutritious, and preferably liquid or semisolid (milk, cereals, broths, fruit-juice, etc.), especially when mastication and deglutition are difficult. Arsenic in the form of Fowler's solution is the remedy *par excellence* in all cases of chorea, except when associated with marked paresis. It should be begun with in ½-drop doses for every year of the child's age, and increased by ½ a drop every other day. Should the urine show the presence of albumin, the lids become puffy, the stomach irritable (pain or nausea), it is advisable to go back to the original dose, or discontinue it entirely for a few days. In the so-called paralytic cases general tonics should be given instead of the arsenic, and in cases with rheumatic or cardiac complications moderate doses of any of the salicylate preparations, with or without the infusion of digitalis—according to indications. During the acme of the disease, the bromids with chloral or a similar hypnotic will be found to act kindly in reducing the choreic movements, allaying the nerve irritability and inducing sleep—which is so essential to the recovery and maintenance of the strength of the patient. In very grave cases chloroform anesthesia may cautiously be resorted to, or lumbar puncture. Finally it is well to remember that chorea is a self-limited disease, and that mild cases usually disappear without polypharmacy—under rest, good food and hydrotherapy.

℞ Liq. potassii arsenitis,
Aq. aurantii flor. .... āā ʒij | 8
M. Sig.: Begin with one drop for every year of the child's age, and increase by one drop every other day, up to full tolerance.

℞ Natrii salicyl.,
Natrii bromidi .... āā ʒiss | 6
Mist. rhei et sodæ.................................. ʒiv | 15
Aq. destil. ....q. s. ad f℥ij | 60
M. Sig.: ʒj every four to six hours for a child 6 years old.

℞ Ferri sulph. ex. .... gr. x | 0.6
Chocolate .... ʒj | 4
M. ft. pulv. no. xx.
Sig.: One powder three times a day for a child 6 years old.

HABIT SPASM.

Children of a nervous temperament quite frequently acquire the habit of spasmodically moving the face (*tic*), fingers and hands, which if not immediately stopped by strict discipline is apt to persist for weeks and months. Habit spasm should not be confounded with chorea.

Lightning-like spasms.

A similar spasmodic condition has been described by Henoch as *"chorea electrica."* It occurs in children from nine to fifteen, in the form of lightning-like spasms, especially of the neck and shoulders, as though produced by a galvanic current. This spasm seems to be identical with "paramyoclonus," but may be hysterical in nature. Electricity does well in these cases.

Suddenly [illegible] in Chorea

SPASMUS NUTANS
(Spasmus Rotatorius; Head Nodding).

In infants.

The disease in question is of obscure origin. It is usually seen in infants of from four to eighteen months of age, chiefly in those suffering from rachitis. The spasmodic movements are generally limited to the muscles innervated by cervical plexus and the accessory nerve, notably the recti capitis, longus colli, scaleni and sternocleidomastoid. In consequence of the irritation the head rotates from side to side or shakes anteroposteriorly at a variably rapid (every second) pace, with occasional interruption, but ceases entirely only during sleep. The head nodding is

Ceases during sleep.

Visual defects.

usually associated with nystagmus and more rarely strabismus or rolling of the eyeballs. In some cases some etiologic relation seems to exist between spasmus nutans and visual disturbance, but whether the defect be in the muscle or nerve supply is still a matter of conjecture.

The spasmodic movements gradually disappear in the course of a few weeks or months, after improvement in the general health.

Differentiation from "juvenile nystagmus."

Spasmus nutans may be confounded with "juvenile congenital nystagmus" (associated with marked visual defects, *e.g.*, disease of the retina, lens, etc.); with brain disease (can readily be recognized by the concomitant symptoms), and epilepsia nutans (*q. v.*).

MIGRAINE; HEMICRANIA

(Sick-headache).

Toxemia.

Reflex irritation.

Migraine is nowadays looked upon as a neurosis, closely allied to epilepsy. It resembles the latter in its periodicity without apparent or definite cause or pathologic organic basis. Cerebral hyperemia or anemia seems to be the immediate cause of an attack. The remote causes are very numerous. Gastrointestinal autointoxication seems to play a prominent rôle, and eye-strain, nasopharyngeal abnormalities, dental caries, helminthiasis, infectious diseases, and general debility are often found to act as predisposing causes. The disease prevails chiefly among nervous children over eight years of age, in girls more frequently than in boys.

Allied to epilepsy.

Attack ends with vomiting.

Like epilepsy, migraine is frequently preceded by premonitory signs, consisting of depression, irritability, visual disturbance, tremor, nausea and vomiting. The child complains of violent headache, usually along half of the head (hemicrania) or occiput. The pain is increased by jars, light, and noises, may last several minutes, hours, or days, and frequently terminates with an attack of vomiting followed by sound sleep, from which the patient awakes very much refreshed and apparently perfectly well. A prolonged attack is not rarely accompanied by mental disturbance and even slight convulsions—in which event it may resemble organic brain disease, *e.g.*, tuberculosis of the brain. The paroxysms may return after weeks, days or months; at all events the disease runs a very chronic course, especially if no energetic efforts are made to determine the underlying cause and to remove it.

Symptomatic treatment.

Where the cause cannot be detected or removed, a great deal of benefit is usually derived from improvement of the general health, especially attention to existing anemia, constipation, etc., and regulation of the diet. Sojourn in the country.

During an attack the patient should be kept quiet in bed, in a dark, well-ventilated room. Local moist heat, and caffeine and quinine (in cerebral anemia), and phenacetin and ergot with sodium bromid (in cerebral hyperemia) are of service to relieve the intense pain.

℞ Natrii bromidi	ʒj	4
Antipyrinæ,		
Caffeinæ natrii benzoatis	āā ʒss	2
Syr. aurantii	q. s. ad f℥ij	60

M. Sig.: ʒj every six hours for a child 6 years old.

PAVOR NOCTURNUS
(Night Terrors).

Night terrors are observed chiefly in nervous children of from three to eight years old. Probably frightened by a horrible dream (seeing of ferocious animals, etc.), the child suddenly awakes, jumps up, sits up or leaves the bed, looks around staringly and anxiously cries or screams for help, or utters incoherent words. After a few minutes he recognizes those about him, quiets down and falls asleep. The attack may recur one or more times a night or at longer intervals, and ultimately disappears (sometimes not until puberty) without serious consequences. In rare instances pavor nocturnus forms a precursor of epilepsy.

Overloaded stomach.

Intestinal worms.

As a rule, pavor nocturnus is brought about by overloading of the stomach before retiring, faulty feeding, hearing of fearful stories or seeing exciting shows; the presence of intestinal worms, adenoids and hypertrophied tonsils, and other local disturbances, and promptly ceases upon removal of the aforementioned causes. The patient should sleep in an airy, slightly illuminated room, on a hard matress, lightly covered and free from tightly fitting nightclothes. The general health should be improved by outdoor air, cod-liver oil, and other tonics. A moderate dose of sodium bromid at bedtime is useful to check frequently recurring attacks.

CHAPTER XV.

Mental Diseases.

THE mental affections of childhood may be classified clinically into (1) those associated with gross and definite physical abnormalities, and (2) those apparently free from such defects. Congenital. The first group in the majority of instances is due to antenatal disease or arrest of development especially of the nerve system, and, hence, is usually in full bloom at a very early age. The second group, as a rule, arises secondarily to traumatism or brain Acquired. disease acquired some time after birth, develops gradually and, therefore, is not demonstrable until the second period of childhood has been reached.

To the first group belong the typical forms of idiocy (hydrocephalic, microcephalic, amaurotic, Mongolian, syphilitic, and paralytic), cretinism and infantilism; the second group embraces imbecility, katatonia, melancholia, mania, and dementia.

IDIOCY AND THE ALLIED MENTAL DEFICIENCIES
(Including Cretinism and Infantilism).

The mental faculties of an infant become appreciative with the Normal mental development. evolution of the senses of hearing and sight. A perfectly normal baby almost fully controls these senses by the end of the third month. As it grows a few months older it begins to show signs of power of attention, perception and memory—it is delighted by bright objects, recognizes familiar faces, more or less appreciates pain or pleasure, etc. When nine months old it is usually capable of understanding certain words spoken to it, and manifests the tendency to imitate sounds and syllables. At about one year of age, if properly trained, strong infants are in full control of the voluntary musculature—creeping, standing, and sometimes walking—and partially so of some of the body functions, such as defecation and urination. Premature infants and those of feeble vitality from other causes may acquire some of these faculties at a much later period of time, and yet grow up to be normal intellectually. However, while no standard time limit can be set down for the establishment of one or the other mental function, failure of their manifestation at an age considerably beyond the supposed normal period, *e.g.*, failure of a child nine months old to indicate a fair possession of the power of attention, or to recognize persons who are in its constant attendance, certainly warrants the suspicion of some mental abnormality.

Abnormal mentality.

In our effort to arrive at a correct conclusion, it is essential to bear in mind the unreliability of parents' views as regards the mental condition of their children—most parents look upon their offspring as the wisest in the land—and the fact that very sharp lines of demarcation cannot always be drawn between infants with normal intelligence and those below the average. Hence, the importance of minute analysis of the family history of the patient; the past history of the patient, particularly as regards nerve disorders and traumatism, and the physical and mental conditions of the child since birth, especially as to their progressive or regressive character.

A nervous heredity being traceable in the great majority of cases of feeble-mindedness, *the family history* is often very enlightening in obscure conditions. Grave neuropsychopathic affections, especially if occurring in the immediate family, demand careful consideration, and a history of dipsomania or syphilis in the parents should always be looked upon with suspicion. Inquiry should be made about the condition of the mother during pregnancy. It has not rarely been demonstrated that infants conceived during convalesence of the mother from prolonged attacks of exhausting diseases (*e.g.*, typhoid) were born idiots. Furthermore, serious domestic troubles, mental anxiety, and physical distress, extreme fright and violent traumatism in the mother may so disturb the normal evolution of the fetal organism as to create grave central disturbances in the offspring. Notice should be taken also of the fact that certain types of mental degeneracy are peculiar to certain races of humanity—often without any discernible cause—as, for example, amaurotic family idiocy in the Hebrew race.

Predisposing factors.

Dipsomania or syphilis in the parents.

Heredity.

The association of feeble-mindedness with any of the aforementioned predisposing factors cannot invariably be accepted as proving the hereditary pathogenesis of the case in question. On the contrary, occasionally totally insane parents beget perfectly sane children, and *vice versâ*. Anticipation of an heredi-

No history [illegible] feeble minded

tary predisposition, therefore, should not deter us from careful scrutiny of the patient's *personal history*, particularly as pertaining to traumatism sustained during birth (compression or fracture of the skull during tardy or instrumental delivery, etc.) or after, and the diseases the child suffered from up to the time of examination.

Traumatism.

No one morbid manifestation in the *past history* of the patient is as corroborative of the latter's abnormal mentality as the occurrence of repeated attacks of convulsions during its early development. Whatever the cause—be it meningitis, gastro-intestinal intoxication, uremia, exanthematous disease or trauma—the very fact that severe convulsions occurred justifies the assumption of some pathologic alteration in the central nerve system, sufficiently grave to predispose to mental impairment. Whenever possible, an attempt should always be made to trace the exact origin of the mental deficiency. Too much stress, however, should not be placed upon the information received. For, at best, histories are only guesswork, unless furnished by very intelligent sources—rather rarely to be expected when dealing with degenerates.

Convulsions.

Having obtained all details as to the patient's family and personal histories, our next, most important, work should be to determine the apparent *mental* and *physical conditions* of the child, especially with the view of comparing them with those of a normally developed child of the same age.

A. Mental Stigmata of Degeneration.

In idiots or mentally backward children the power of attention is either very poorly developed or entirely absent. They fail to take notice of their surroundings, stare vaguely into empty space, or move the eyes irregularly in all directions, apparently seeing nothing. This want of attention may be due to partial or total blindness of central origin without involvement of the eyeball, as is frequently the case in amaurotic family idiocy. Occasionally the patient may be subject to congenital cataract, microphthalmos, coloboma iridis, irideremia, lesions of the vitreous, strabismus, and similar visual defects which in conjunction with other stigmata facilitate the diagnosis.

Lack of power of attention.

Except in encephalitic or amaurotic family idiocy, defective hearing of central origin is rather uncommon. On the other

Defective hearing.

hand, deafness is not rarely observed as a result of congenital malformation or acquired disease of the auditory canal. In the latter event a history of normal hearing at an earlier age is usually obtainable.

Voracious appetite.

Genuine idiocy is invariably associated with a voracious appetite, and, owing to imperfect development of the senses of taste and smell, no choice is manifested as to the kind of food given. Everything that comes along is rapidly devoured; hence the frequency of gastrointestinal disturbances in feeble-minded children. The sense of smell is sometimes so obtuse that even irritating odors are not productive of local or reflex phenomena in the respiratory tract. On the other hand, occasionally idiots, like animals, are endowed with a hyperacute sense of smell.

Obtuse senses of touch, pain, heat or cold.

Most idiots are insensible to touch, pain, heat or cold—anomalies of sensibility which explain the frequency with which such children are subjected to external injuries and voluntary bodily mutilations. In some of them, however, especially in those with marked defective vision, tactile sense is so highly developed that by this means alone they are able to recognize persons who feed and care for them.

Deaf-mutism.

Congenital deafness is, of course, associated with mutism. But even where hearing is intact, few idiots are able to speak. Some of them, by imitation, do learn to utter a few words, but their expressions usually bear no relation to any distinct desire or action, and they understand words spoken to them no better than what they speak. Moreover, their power of imitation is very much delayed in development or may never become manifest—all depending upon their poor faculty of attention. In partial idiocy, such as mild forms of infantilism, cretinism, encephalitis, or microcephalus, the power of conversation may reach a certain degree of potency, but their vocabulary is usually very limited and fragmentary.

This form of *pseudo*-deaf-mutism, like genuine deaf-mutism, is not necessarily indicative of the degree of intellectual development of the idiotic children in question. Some idiots, *e.g.*, microcephalics, may incoherently chatter for hours, and yet be no wiser than those who never utter a single word.

Lack of reasoning power.

With few exceptions, idiots are unable to acquire, retain, associate or evolve ideas; reason, judge, or appreciate their personality, their actions or their surroundings. Instinctively they may cling to those who feed and take care of them, like animals obey their masters, after prolonged training perform certain actions, and even manifest a certain degree of reasoning power. In the majority of instances, however, their performances are mechanical and automatic. They may for hours lie or sit in one position and indulge in certain movements, without by attitude or expression indicating the desire for a change, or even betraying any discomfort previous to or after the acts of defecation or urination.

Amaurotic—totally devoid of reason.

Microcephalic—vulgar, brutal.

Hydrocephalic—gentle, timid.

Paralytic—fairly intelligent.

Mongolian—improves with age.

Cretin—improves under thyroid treatment; otherwise childish.

As compared with the different varieties of idiocy, intelligence is least allotted to the amaurotic idiot. Deprived of sight and hearing since early infancy, limp and languid as a result of the ever increasing atony of its musculature, the helpless creature gradually loses all its senses, and, fortunately, also its life. Microcephiles are nearly as badly endowed with intelligence as the former group, but their stupidity is not progressive in character. As they grow older they are able to feed themselves, and with hearing intact they may learn to talk—talk without sequence or measure. Genuine microcephalic idiots are obstinate, vulgar, and brutal. Almost the exact opposite characteristics are observed in hydrocephalic idiots. They are ordinarily soft, gentle, timid, sorrowful, but little impressionable or curious. As very marked cases of hydrocephalus usually succumb at birth or soon after, those surviving usually possess a greater degree of intelligence than microcephiles, nay, at times they may grow up to be perfectly normal. The mental impairment following meningitis, or encephalitis, varies with the extent of the brain lesion produced by the inflammatory process. Deaf-mutism, aphasias and amaurosis being common sequelæ, little intellectual capacity can be expected. In the absence of these defects, the little patients may gradually acquire a fair measure of intelligence. The same observations practically hold good for syphilitic idiocy. Tics and convulsions are not rare in both of these types of idiocy. The Mongolian idiot is a restless creature. Totally idiotic in early infancy, he gradually shows signs of improving mentality. He learns to appreciate his surroundings, and to make himself understood by a language of his own. He learns to run about at an earlier age than most other idiots, and not rarely shows destructive tendencies. Under suitable treatment cretins often attain a fair measure of intellectual development. Like hydrocephiles they are timid, gentle, and unassuming. They retain their childish tastes for a long time, and sometimes for life, if left untreated. In the latter respects infantilism differs little from cretinism. In infantilism, however, there is much greater control of the muscular system, and quicker response to medication and training.

Infantilism—responds to education.

Significant as the aforementioned mental stigmata of degeneration are to disclose the existence of idiocy as a whole, they can rarely be relied upon in the determination of the exact form of the affection. For this purpose the physical peculiarities of idiots presently to be related are almost invariably decisive.

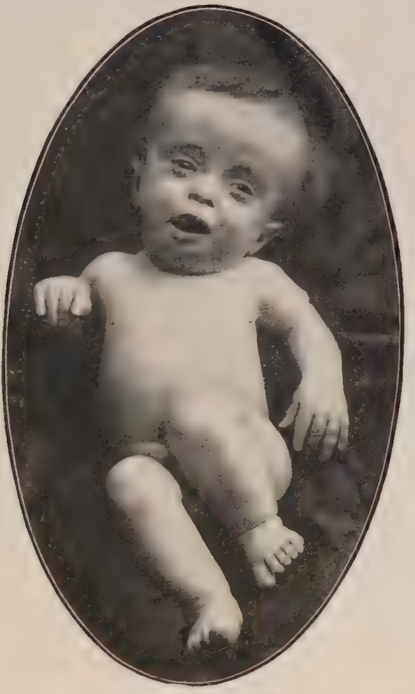

Fig. 185.—Hydrocephalic Idiot. (*Sheffield.*)

B. Physical Stigmata of Degeneration.

Hydrocephalic—large, soft head.

The cranium of a hydrocephalic[1] (see Fig. 185) is large, ball-shaped, and its circumference widest at the temples. It contrasts strongly with the small, delicate face. The fontanelles are separated, the eyebrows are scarcely indicated, and the mouth and nose are small. In microcephalus[2] (see Fig. 186) the head

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[1] See page 124. [2] See page 123.

Microcephalic—small head, prematurely ossified.

Syphilitic—head irregularly bossed; Hutchinson teeth.

Mongolian—short, pug-nose; prominent cheek bones.

Amaurotic—head thrown forward or backward.

is small, narrow anteriorly and devoid of posterior projection. It greatly resembles that of an animal. The fontanelles are prematurely ossified. The eyes are small, the ears project, and the nose and the lower jaw are large. In syphilitic idiots the head is unevenly enlarged. The skull is irregularly bossed and traversed by prominent blue veins. The nose is often saddle-shaped. The upper central incisors are notched. The lips are usually thick and the angles of the mouth not rarely marked by rhagades. The cranium of the Mongolian (Figs. 188, 189) idiot is somewhat smaller than normal, rounded, with the occiput running quite parallel with the plane of the face. The face is sunken, the nose short and broad and bound laterally toward the eyes by distinct vertical folds. The cheek bones are prominent, and the tongue protrudes. No characteristic physical signs of degeneration are apparent on the cranium of the amaurotic idiot[1] (see Fig. 187), except that, owing to the general atony of the musculature, the patient is unable to hold up his head.

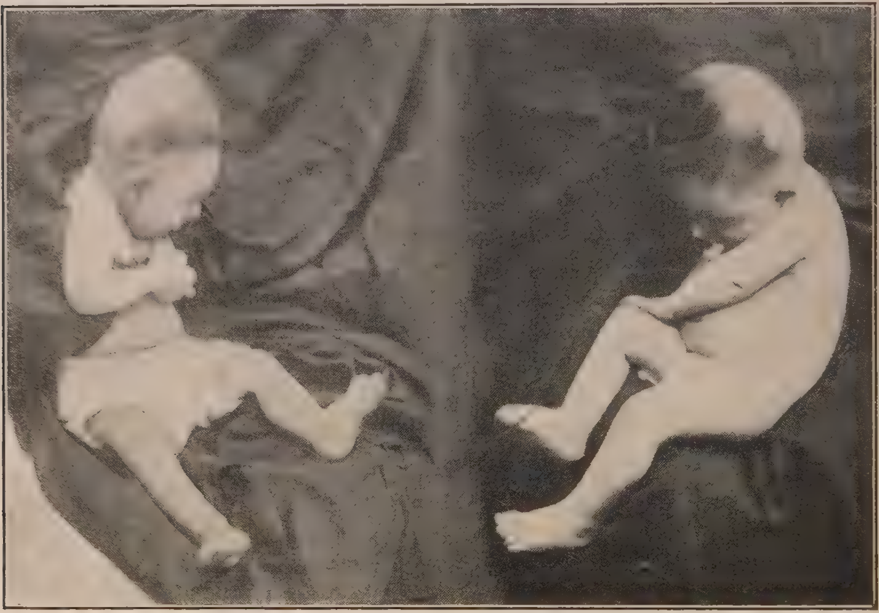

Fig. 186.—Microcephalic Idiot. Fig. 187.—Amaurotic Idiot.

STATUS IDIOTICUS
(peculiar attitude assumed by idiots in sitting posture). (*Sheffield.*)

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[1] See page 583.

The face is delicate. The skull of the cretin[1] (see Fig. 190) is rather larger than normal, sparsely covered by thin, lusterless hair, and set upon a thick, short neck. The face is weak and senile, the eyelids and lips are thick, the tongue is heavy and often protrudes from the half closed mouth. Meningitic, encephalitic, or paralytic idiocy (see Fig. 191) usually presents no characteristic cranial physical signs, except when due to severe traumatism at birth or after. In some congenital cases there is marked flattening of the temporal bone of one side corresponding with the lesion in the brain (porencephalia, etc.). In infantilism (Figs. 192, 193) the skull is smaller than normal, and the face is either plump and senile (typus Brissaud), or thin, delicate and infantile (typus Lorain).

Cretinic—puffy face; protruding tongue.

Paralytic—involvement of cranial nerves; porencephalia.

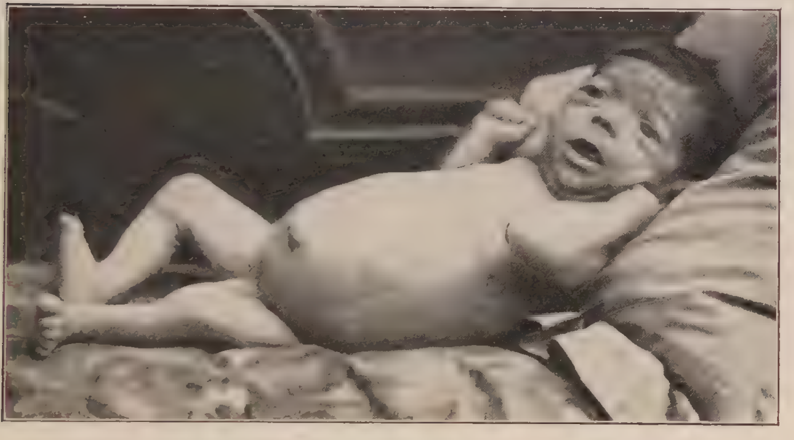

Fig. 188.—Mongolian Idiocy. (Calmuck type.) (*Sheffield.*)

Infantilism—small head; senile face.

The teeth of the great majority of idiotic children are irregularly implanted, faulty in form, and excessive or deficient in number. Owing to irregularity of the dental arches and size of the teeth, the patient is frequently unable to close his mouth—which should not be mistaken for the open mouth associated with nasal obstruction—and the constantly dribbling saliva not rarely leads to painful excoriation of the chin. The lips are often congenitally malformed. The palate is high and narrow and quite frequently clefted. Internal inspection of the nose usually reveals numerous deviations from the normal construction. The ears often project or are asymmetrical. Idiots

Teeth irregularly implanted.

Palate high and narrow.

Ears asymmetrical.

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[1] See page 488.

Vision defective. often present divers anomalies of the eyes varying from simple errors of refraction to total absence of the eyes.

Except the presence of pseudolipomatous masses in the myxedematous, there are no pathognomonic physical peculiarities of the trunk which may be helpful in the differential diagnosis between the numerous forms of mental backwardness.

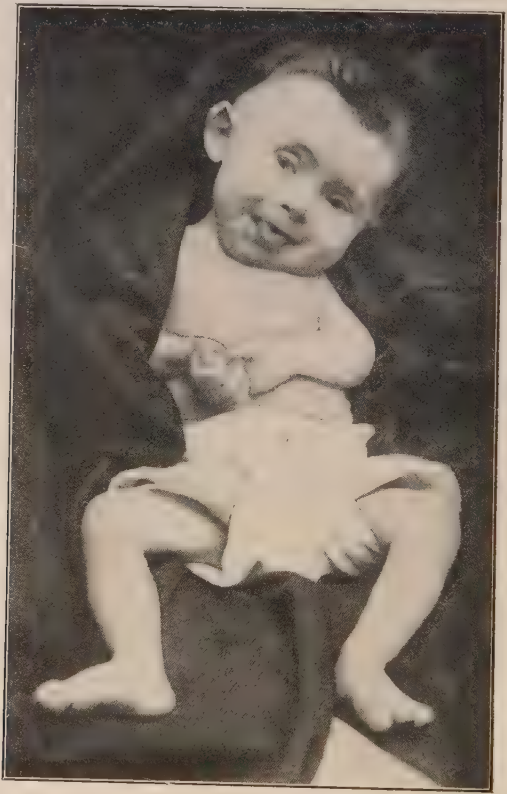

Fig. 189.—Mongolian Idiocy. (Malay type.) (*Sheffield.*)

Thorax deformed. Most idiots are undersized, present more or less marked deformities of the thorax and spine, large abdomen, hernias and narrow Hernia. pelves. The genitalia are often undeveloped and malformed.

Hydrocephalic—paraplegia. The condition of the extremities varies in the different types of idiocy. The hydrocephile often suffers from paraplegia with spastic rigidity of the muscles, and is thus unable either to walk or stand. The upper extremities are usually normal, and only occasionally affected by contractures and athetotic movements. The microcephalic idiot is an extremely restless creature and rarely sits or stands still even if supported. Some few of them are completely rigid, and others, when they grow older, manage to walk about. The syphilitic, as a rule, are helpless, principally as a result of deformities of the extremities and rarefying and softening inflammatory processes at the articulations. The child walks as if paralyzed, if it can walk at all. The

Micro-cephalic—Rigidity.

Syphilitic—pseudo-paralysis.

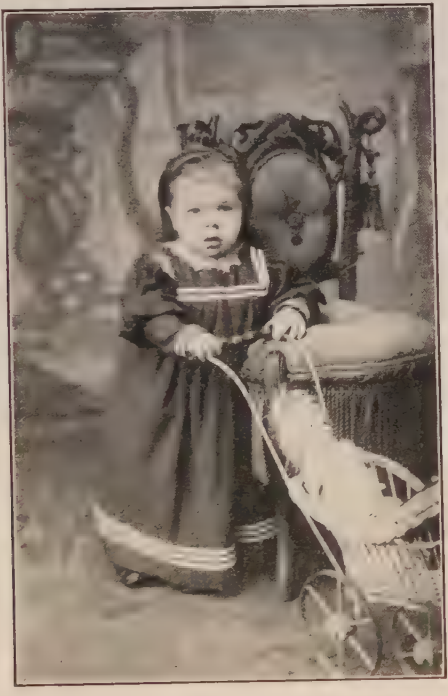

Fig. 190.—Cretinic Idiot (8 years old). Note infantile appearance and tastes. (*Sheffield.*)

Mongolian idiot begins to walk at a much later age than the normal baby. His joints are weak, and his hands and feet plump. Amaurotic idiocy is characterized by muscular atony which gradually terminates in general paralysis. The cretin has a peculiar dragging and awkward gait, and often presents deformities of the extremities and thickening of the joints. Unilateral or bilateral hemiplegia with contracture and athetosis is pathognomonic of the paralytic idiot. Where one hemisphere is involved he has a tottering gait. In infantilism the extremities

Mongolian—plump extremities; awkward gait.

Cretinic—dragging gait.

Paralytic—Hemi- or di-plegia.

are apparently normal in shape, but their musculature is weak and flabby. A radiogram (see Figs. 194, 195) of the hand usually reveals backward development of the centers of ossification of the carpal bones and of the epiphyses of the metacarpals and phalanges.

Backward development of carpal bones.

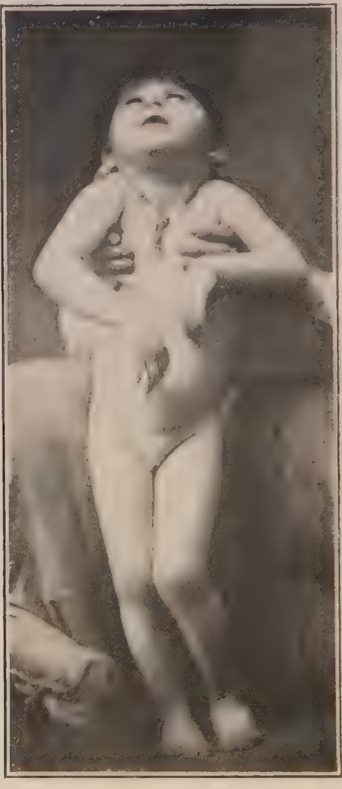

Fig. 191.—Paralytic Idiot (following obstetric cerebral paralysis). (*Sheffield.*)

In addition to the aforementioned stigmata of degeneration idiots often present asymmetry, malformation, superabundance or deficiency of fingers and toes (see Fig. 193), club-foot, ankyloses, dislocation of the extremities, diastases (see diastasis recti abdominis, Fig. 37), and, as a result of lesions in the brain and spinal cord, divers forms of paralyses and contractures, and local and general atrophies, etc.

Deformities of fingers.

With due appreciation of the mental and physical peculiarities of the various forms of idiocy, there is rarely any difficulty in

Importance of early diagnosis.

arriving at a correct diagnosis. The importance of early individualization cannot be too strongly emphasized, for, with a full knowledge of the type of idiocy a great deal can often be accomplished in the way of treatment. I am referring especially to the phenomenal success obtained from the administration of thyroid gland in cretinism (*q. v.*), and the great improvement in milder forms of idiocy (*imbecility*) that results from systematic methods of training and education. It is greatly to be regretted that the laity and the physician alike are so little interested in the humanitarian problem of providing suitable training schools for the amelioration of the unfortunate condition of the idiot and mentally defected.] [illegible]

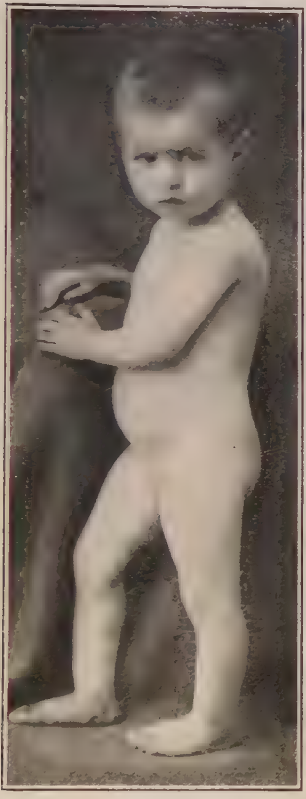

Fig. 192.—Infantilism. (Typus Brissaud.) Child six years old, 32 inches in height. (*Sheffield.*)

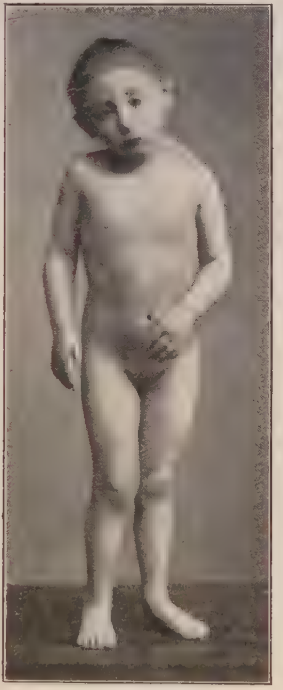

Fig. 193.—Infantilism. (Typus Lorain.) Five years old, 31 inches in height. Acts like two-year-old baby. Note absence of left thumb and rudimentary development of right thumb. (*Sheffield.*)

Thyroid treatment.

Training.

Education.

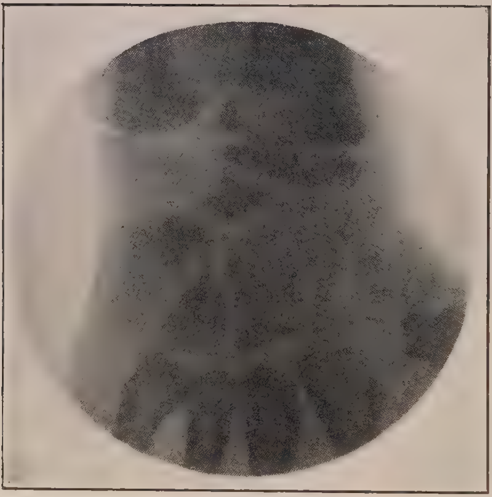

Fig. 194.—Skiagram of Wrist of Normal Child Six Years Old. Note greater number of carpi than in idiotic wrist. (*Sheffield.*)

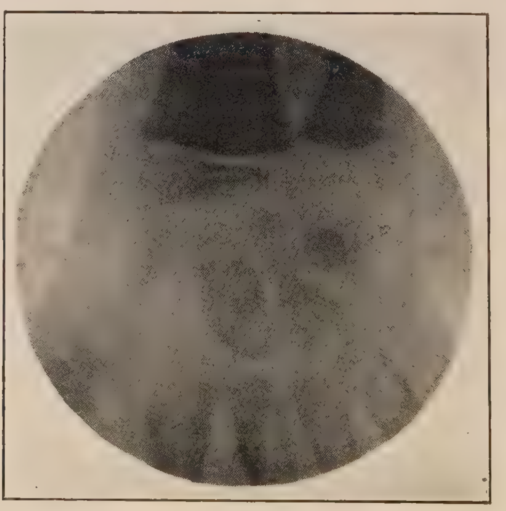

Fig. 195.—Skiagram of Wrist of Idiot Ten Years Old. Note absence of several carpi. (*Sheffield.*)

For details of treatment of hydrocephalus, microcephalus, cretinism, etc., the reader is referred to the respective chapters on the subjects in question.

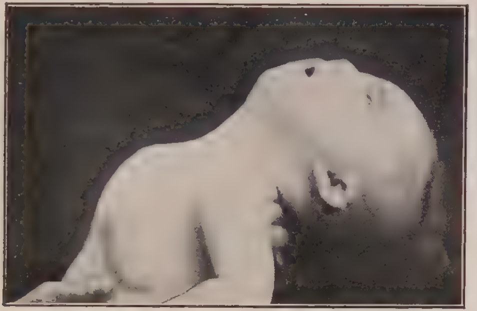

Fig. 196.—Amaurotic Family Idiocy (14 months old). Note inability to hold up head. (*Sheffield.*)

AMAUROTIC FAMILY IDIOCY.[1]

This type of idiocy occurs in several members of the same family and shows a predilection for those of the Hebrew race.

Arrested development.

Its etiology is still obscure. While all observers agree that it is due to arrested development and sequential degeneration first of the gray matter and later of the white fibers of the brain and cord, it is still undecided whether the degeneration is of antenatal or postnatal (toxemia) origin. The very recent

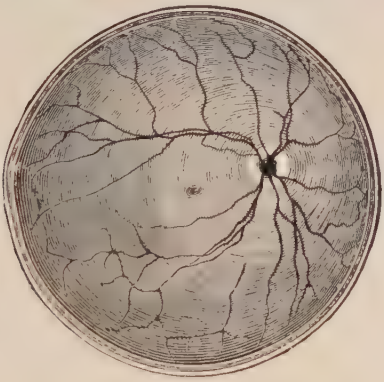

Fig. 197.—The Normal Fundus of the Right Eye. (*Henle.*)

Toxemia.

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[1] To B. Sachs, of New York, we are indebted for the name and most of the knowledge of the disease in question.

observation that there is also a "late" form of this disease seems to point in favor of its being acquired.

However this may be, the symptom-complex is very charac-
Normal at birth. teristic. The normally born infant which seems to thrive fairly well up to about 4 or 8 months begins to show symptoms of debility and atony of the entire musculature. As it grows older, the mental development, instead of progressing normally, fails to come up to the normal standard, so that it soon becomes evident that the infant is absolutely idiotic. Further examination reveals that the child sees very little or not at all (due to degeneration of the papilla and optic neuritis; there is a grayish-white cloudiness around the cherry-red macula), and that its hearing, while at first hyperacute, gradually becomes obtuse. The optic atrophy and general paresis are usually complete when the child is 1 year old. At a later stage of the affection there are also nystagmus, strabismus and "explosive laughter."

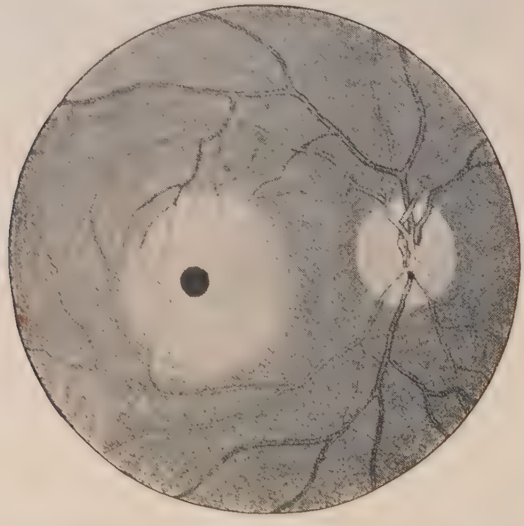

Fig. 198.—Macular Change [cherry-red color] in Amaurotic Family Idiocy. (*Tay.*)

Blindness.

Defective hearing.

With careful attention—fresh air; good food; care of the skin; frequent change of position to prevent hypostatic pneumonia —the patients may continue to exist up to about 2 years old; without suitable care, however, they usually succumb at an earlier period from marasmus and exhaustion, the latter often as a result of inanition subsequent to difficult deglutition.

Fatal.

For the differential diagnosis between amaurotic and other forms of idiocy, see page 570.

IMBECILITY
(Feeble-mindedness).

Inherent mental privation.

Imbecility is closely related to idiocy, and is based upon some inherent mental privation which no amount of education can entirely overcome. The condition is usually not detected until the child goes to school, when it is found that as compared with the normal pupil he is backward in understanding and reasoning, though he may be singularly developed in special directions, *e.g.*, memory, mechanical aptitude. Further observation reveals also that the imbecile is exceedingly emotional, easily irritated and appeased with difficulty, shows an irresistible passion to lie, steal and play truant, and that long before maturity his sexual inclinations are in the highest state of depravity. His moral decrepitude increases from year to year, and may range from theft, arson and rape to homicide and suicide.

Moral decrepitude.

Imbeciles should be placed under the control of experienced pedagogues, preferably in some lonely country place.

KATATONIA
(Hebephrenia).

This mental affection is usually encountered in children over ten years of age, and especially in girls at the period of puberty. It usually begins with a prodromic stage of depression and apathy during which the child loses interest in her school work, and complains of divers imaginary ailments. This stage is soon followed by one of anxiety and hallucinations or outbreaks of emotional excitement, silly and hilarious in nature. As the disease advances, the condition is often complicated by manifestations of mania with a marked tendency to destructiveness and violence, occasionally also by attacks of stupor, catalepsy, affections of speech, refusal of food, convulsive movements, etc.

Depression.

Hallucinations.

Mania.

In favorable cases the mental disturbance gradually subsides within a few weeks or months, often leaving behind symptoms of imbecility. In unfavorable cases the disease passes into a state of total idiocy. Little, if anything, can be done to influence the course of the affection.

MELANCHOLIA.

Depression.

Depression of spirits is not rarely observed in children from ten to fifteen years of age and sometimes even in younger ones. The child refuses to play, laments and cries, broods over imaginary wrong acts, and occasionally falls into paroxysms of rage. Melancholia not rarely leads to attempts of self-destruction.

Tendency to self-destruction.

The prognosis of this affection is fairly favorable (after weeks or months); some cases, however, may proceed to mania or even dementia. Rest and good food are essential in the treatment.

MANIA.

Physical and mental hyper-activity.

In contrast to melancholia mania is characterized by acceleration of every physical and mental activity. Thoughts and impulses follow one another with unusual rapidity. The patient talks, rages, screams and tries to destroy everything in sight. He also suffers from hallucinations and delusions of greatness. While mania often ends in recovery after from six to twelve months, it also shows a great tendency to recurrence or to alternate with attacks of melancholia—*circular insanity*—in which event the prognosis is very bad.

Recurrences.

The treatment, in addition to rest and proper nourishment, is symptomatic—calming of the excitement by means of hyoscine hydrobromate.

DEMENTIA.

Acute dementia is rare in children under twelve years of age. It is apt to follow severe infectious diseases, such as typhoid fever or scarlatina, or sudden shock and mental and physical overexertion. It is manifested by gradual weakening of the mind, characterized by loss of memory, lack of power of attention, interest, and curiosity and tendency to stupor. After weeks or months of rest, ample nutrition and tonic treatment there is usually a progressive return of intellect and gradual recovery. More rarely it terminates in permanent weak-mindedness.

Loss of memory; stupor.

Dementia *paralytica,* which is very uncommon in children and usually based upon hereditary syphilis, presents identical symptoms as in adults. Thus, tremor, slurring speech, pupillary inequality, ataxia, trophic changes, and paresis; gradual loss of intellect with development of unsystematized ideas of self-importance. The course of this form of dementia is chronic (several

Paralysis; tremor; slurring speech.

years) and invariably ends fatally. Slight improvement may occasionally be obtained from cautious use of mercury and the iodids.

HYSTERIA.

Hysteria is a neuropsychosis, a product of faulty environment and education.[1] It is rare in children under eight years of age, but quite common in older ones, and especially in girls.

Shock; trauma.

The onset of hysteria can frequently be traced to some sudden violent emotion (shock) with, or, more rarely, without bodily injury. The attendant circumstances at the time of the psychic disturbance often serves to determine the seat of the hysterical lesion, *e.g.*, hysterical deafness after explosion, paralysis or contracture of an extremity, after a trivial injury.

The symptomatology of hysteria is characteristic for its multiplicity and mutability. It may closely simulate that of any organic disease, but its spuriousness can usually be detected after careful scrutiny. The diagnostic perplexities augment, however, with accidental concurrence of some acute affection or preexistence of a chronic organic disease.

Paralysis of extremities.

Paralysis of the extremities with or without contracture forms a frequent hysterical manifestation. It may appear in the form of paraplegia, monoplegia or hemiplegia, and thus resemble myelitis, poliomyelitis or cerebral paralysis. In hysterical "spinal" paralysis, however, there is rarely absolute loss of muscular power. Muscular atrophy is absent or slight, and electric irritability remains normal. In hysterical "cerebral" paralysis also the loss of power is rarely complete and the leg is often more affected than the arm. The face usually remains uninvolved. A peculiar form of either continuous or intermittent pseudoparalysis is occasionally met in children, which has been described by Blocq as astasia-abasia. In this condition the muscles of the lower extremities can be freely used except in standing or walking. If the latter is attempted, the patient immediately falls to the ground or begins to tremble and topples over, or manifests ataxic symptoms (*cerebellar* type). The difficulty in walking is sometimes overcome after a few steps are taken.

Differentiation from spinal and cerebral paralysis.

Astasia-abasia.

Contractures.

The hysterical contractures may involve the articulations, groups of muscles or a part of a muscle. As a rule, the joints of the tapering extremities are most frequently affected. All sorts of deformities may arise which may greatly resemble genuine joint and bone disease (*e.g.*, hip-joint disease, spondylitis, talipes, etc.), and lead to errors in the diagnosis. The more sudden onset, the irregularity of its course, the tendency to change its situation, and the concomitance of other evidences of hysteria, all help the exclusion of organic disease. At a later stage the diagnosis of hysterical contracture can frequently be made by the absence of local thickening, or active inflammation of the bone or muscle and its disappearance under anesthesia. Where a part of a muscle is affected the contracture may give rise to circumscribed swellings. Allied in nature are also the so-called "phantom tumors" occasionally observed on the lower portion of the abdomen, and the peculiar "ballooning" of the hypogastrium manifested with each expiration. Occasionally the abdominal enlargement is general and not rarely accompanied by local tenderness. Furthermore, the hysterical tympanites may be associated with vomiting, anorexia, singultus, disturbed respiration, retention of the urine, etc., and thus give rise to the clinical picture of peritonitis which may test the skill of even the best diagnostician. As a rule, obstipation and fever are absent in these cases and the vomitus is not so persistent as in true peritonitis. Of course, vomiting, anorexia, tachypnea, etc., may exist independently of the hysteria and greatly obscure the diagnosis.

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[1] "A Contribution to the Study of Hysteria in Children," by Dr. H. B. Sheffield, New York Medical Journal, September 17 and 24, 1898.

Differentiation from inflammatory deformities.

Phantom tumor.

Ballooning.

Differentiation from peritonitis.

The symptoms thus far enumerated represent principally the neurotic element of hysteria. To those may be added the occasionally occurring cataleptic states, spasm of the laryngeal muscles (croup), dysphagia, aphonia, aphasia with spells of coughing, singing or stuttering, asthma, amblyopia, hemianopsia, contraction of the visual field, amaurosis and blepharospasm.

Catalepsy.

Amaurosis.

In another group of cases the psychic element predominates. Here too, however, there is generally a great display of spasmodic and convulsive movements ranging between simple or choreic tremor to marked epileptiform convulsions (hysteroepilepsy). The movements may assume the form of athletic exercises, such as rowing, swimming, punching, etc.—*chorea rhythmica;* or the patient may act as though possessed, climb walls, turn somersaults, and perform all sorts of stunts—*chorea magna.* Still more advanced cases of hysteria may be manifested by attacks of sopor, night terrors, somnambulism, hallucinations, delirium and mania.

Psychic disturbances.

Chorea rhythmica.

Chorea magna.

Hystero-epilepsy.

Hysteroepilepsy is comparatively rare in children. An attack is usually preceded by emotional excitement, globus hystericus, etc., and may be induced by pressure upon sensitive areas—hysterogenic zones—of the body, such as the hypochondriac or spinal regions. Hysteroepilepsy differs from genuine epilepsy in the following respects:—

Differentiation from genuine epilepsy.

EPILEPSY.	HYSTEROEPILEPSY.
Onset sudden.	Preceded by emotional excitement.
Consciousness entirely lost.	Partially preserved.
Convulsions pre-eminently clonic.	Tumultuous, accompanied by moaning, screaming, crying, etc.
Duration short, followed by stupor.	Longer; followed by restlessness.

Hysteria generally proceeds a very chronic course, with temporary improvement and relapses. Of course, it very much depends upon the etiologic factors, the time when treatment is begun and the energy with which it is carried out.

Usually acquired by imitation.

Without denying the transmissibility from parent to offspring of a certain degree of nerve instability which may predispose to hysteria, in the great majority of instances this disease is acquired as a result of harmful influence of faulty environment and education. A child repeatedly seeing its mother, for example, in a state of emotional excitement or frenzy, sooner or later, consciously and deliberately, or otherwise, learns to imitate its mother's hysterical performances, the habit of imitation gradually leading to aberration of the normal cerebral functions. Unable as the mother is to control her own abnormal actions and feelings, she can hardly be equal to the occasion to guide her children in the right direction. On the contrary, the child is allowed to have its own way, is made the central figure of the household, and spoiled by overtenderness. If in addition such methods of education are adopted as will overtax the child's mental capacity (*e.g.*, the study of music, painting, emotional recitations, etc., in addition to arduous school work), a deranged state of mind sooner or later supervenes which is most susceptible to the aforementioned pernicious influences. Less potent factors in the predisposition to hysteria are: The use of alcoholic beverages, acute infectious diseases, prolonged disturbance of the sexual (masturbation!), digestive and circulatory (anemia) systems, in fact, anything that will undermine the physical or mental condition of the child.

Other predisposing causes.

With these principal etiologic factors in view the indications for the treatment of hysteria in children are self evident. The

Discipline. Outdoor life. Suitable nutrition.

patient should be removed from the hysterical environment, and placed under the care of one who with kindness but firmness can control its destiny. Change of residence from the noisy city to the restful country often works wonders. The child should lead an outdoor life, and every effort should be made to raise its general bodily development. The food should be ample and nutritious, free from alcoholic beverages. Milk foods should be given preference to meats. The education should be restricted to the simplest school work, or, for a time at least, entirely suspended.

Hydrotherapy. Suggestion. Isolation.

The active treatment of hysteria is essentially symptomatic. Warm baths and cold showers and general massage are useful in all cases. Paralyses and contractures frequently yield to electricity, its action being probably suggestive in nature. Suggestion by electricity or other spectacular procedures is also effective in relieving local conditions, such as aphonia, stuttering, blindness, and the like. Hysteroepilepsy and maniacal outbreaks call for isolation, rest in bed and the administration of small doses of the bromids and valaria. Disregard of the patient's complaints and severity will often cure all sorts of hysterical phenomena where kinder therapeutic measures ordinarily fail.

℞ Ext. humuli fl.	ʒiij	12
Infusi valerianæ,		
Aq. aurantii flor.	āā ℥j	30

M. Sig.: ʒj every four hours for a child 10 years old.

CHAPTER XVI.

Diseases of the Skin.

SKIN affections of children like those of adults may be classified into systemic and local. To the former class belong chiefly the large group of exanthemata; the rashes arising as a result of faulty metabolism and autointoxication, including the different forms of purpura, erythema and drug eruptions; the syphilides and tuberculous lesions and the obscure dermatoneuroses. The local skin diseases embrace the local parasitic affections, the lesions following mechanic, traumatic, thermic and chemic irritations.

Systemic.

Local.

As the greater number of morbid skin manifestations have received due consideration in connection with the underlying diseases, we shall here limit our discussion to the skin eruptions which yield principally to local treatment.

ECZEMA.

Eczema in children is usually observed in subacute or chronic form. It ordinarily begins with localized, more rarely diffuse, redness of the skin, slight edema, burning and itching. The condition is soon aggravated by the appearance of papules, vesicles and pustules, and, if not promptly responding to treatment, by scabs, scales and fissures.

Redness; edema; burning and itching.

Eczema may remain localized, especially on the face and head, or become generalized. Eczema of the face and head is usually seen in young infants, and is very refractory to treatment. In its typical form, the eruption of eczema faciei is generally spoken of as "crusta lactea," and consists of more or less coherent scabs of greenish or blackish-brown color, here and there interrupted by areas of red, moist ("weeping surface"), and excoriated skin. From the face the eruption usually extends to the forehead, ears and head (eczema or seborrhea capitis). After prolonged duration the hair loses its luster, becomes thin and short and the adjacent glands are painful and swollen, and often the seat

Crusta lactea.

Adenitis.

of a pustular eruption as a result of scratching and secondary infection.

Chronic course.

The course of eczema is very tedious. It may last weeks, months, or years. Improvement often alternates with aggravation of the condition. This is true especially of eczema accompanying constitutional derangement, *e.g.*, gastrointestinal intoxication. The duration of the disease is often greatly prolonged by infection of the diseased as well as healthy areas with divers parasites during the act of scratching.

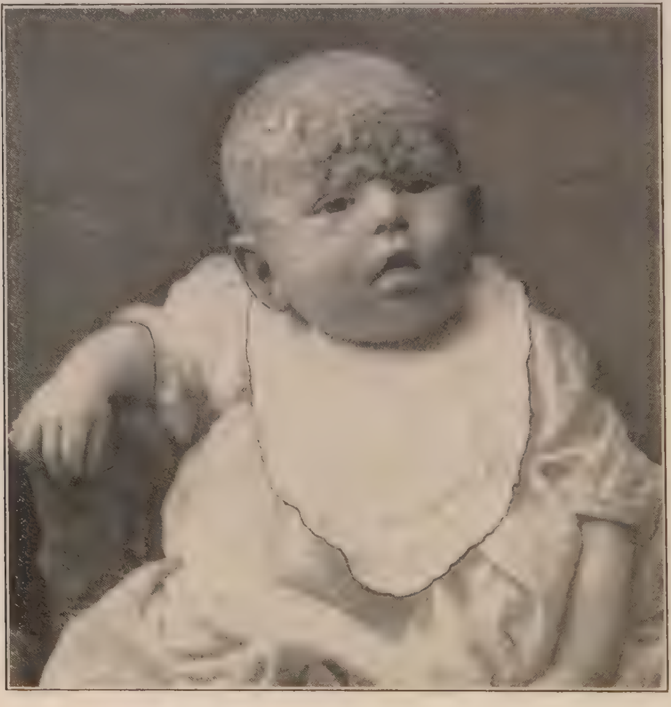

Fig. 199.—Seborrheic Eczema of Head and Face. (*Sheffield.*)

Secondary infection.

Avoid irritation.

The success in the management of eczema depends greatly upon the ease with which the underlying causes can be prevented or removed. The infantile skin being very delicate and vulnerable, it is essential to avoid its undue exposure to mechanical (scratching; woolen, rough underwear, etc.), thermal (excessive heat or cold, also direct action of the sun, etc.) and chemical (rubefacients, irritating soaps, urine, acrid discharges, etc.)

Correct faulty diet.

irritation. The diet should be bland (not too rich in fat), and regulated as to the time of feeding and its quantity. Constipation should be promptly remedied. Cleanliness of the skin and everything coming in contact with it should be insured.

Avoid excessive moisture.

The active treatment of eczema should be regulated in accord with the stage of the disease. While the skin is highly inflamed, all sorts of irritation should be interdicted. Tub-bathing of the entire body should be discontinued for a time, firstly, because of the tendency of water to irritate the denuded skin, and, secondly, in view of the possibility—particularly in eczema due to external parasitic infection—of conveying the disease from one portion of the skin to the other. The healthy parts of the body, however, should be kept scrupulously clean by frequent sponging, followed by careful drying.

Protective ointments.

The following soothing and protective ointment, employed with great success at the New York Post-graduate Hospital, will be found invaluable in the great majority of acute or subacute cases:—

℞ Zinci oxidi,
Pulveris cretæ ....āā ʒiv | 16
Mix, and add with constant stirring:
Olei lini (hot),
Liq. plumbi subacet. dil. ....āā ʒij | 8

Prevent scratching.

The ointment is applied once or twice a day thickly over the affected areas and covered by sterile gauze held in place by means of a bandage. Scratching of the skin should be prevented by mechanical means, such as celluloid armlets, and the like. Excoriated surfaces often heal promptly after painting with a 2 per cent. solution of nitrate of silver.

Stimulating ointments.

After the inflammation subsides and scales and crusts firmly adhere to the skin, the soothing ointments are gradually replaced by those of a stimulating nature. The crusts are softened with carbolized oil (1 to 100), and gently removed. The hairy portions of the body are carefully shaved and cleansed with carbolized oil. After giving the affected skin a few hours' rest we apply one of the following preparations:—

℞ Acidi salicylici,
Bismuthi subgal. ....āā gr. xx | 1.3
Thymolis .... gr. v | 0.3
Pulveris amyli .... ʒiij | 12
Ung. hydrargyri ammoniati .... ʒij | 8
Ung. zinci oxidi ....q. s. ad ℥ij | 60

℞ Resorcini	gr. xx	1.3
Acidi carbolici	gr. x	0.65
Olei cadini	*m* xx	1.3
Sulphuris precipitatis	ʒij	8
Ung. petrolati	q. s. ad ℥ij	60

Intestinal irrigation.

High intestinal irrigation once a day with a quart or two of plain water or with the addition of 2 per cent. of bicarbonate of soda is useful in all cases. In gastric hyperacidity carbonate of magnesium (gr. xxx, once a day) acts well. Obese children suffering from obstinate eczema with dryness of the skin often do well on minute doses of thyroid extract. Finally, it is worth remembering that protracted eczema is occasionally a manifestation of hereditary syphilis, and responds promptly to the exhibition of mercury and the iodids.

URTICARIA

(Hives, Nettle Rash).

Transient multiform eruption.

"Wheals."

Urticaria is characterized by a multiform eruption of whitish, pinkish or reddish color upon different portions of the body, which is sudden in appearance and disappearance, and shows a tendency to repeated recurrences. The eruption may consist of circular or spiral elevations ("wheals"), papules, vesicles, or hemorrhagic spots, and is generally associated with intense itching and stinging. It is frequently preceded and accompanied by gastric and nervous disturbances and rise of temperature.

Tendency to prurigo.

Recurrent urticaria is prone to leave behind marked pigmentation of the skin or to terminate into *prurigo,* a very chronic skin affection manifested by dryness, hypertrophy and pigmentation of the skin and inflammation of the neighboring glands.

Faulty feeding.

As in the majority of instances, urticaria in children is the result of faulty feeding, especially of eating candies and cakes of poor quality, fish, fresh berries and the like, it is essential promptly to regulate the diet, and to clear the gastrointestinal tract of the obnoxious material. The latter is best accomplished by small doses of calomel, magnesium carbonate and sodium bicarbonate and a high enema. To relieve itching we may resort to warm baths with bicarbonate of soda (½ to 1 pound); sponging of the body with vinegar followed by glycerin, or to the following preparations:—

℞ Thymolis	gr. v to x	0.3 to 0.65
Ung. aquæ rosæ	℥j	30

Sig.: P. r. n.

℞ Aquæ ammoniæ	ʒss	2
Aquæ hamamelidis	℥iij	90

Sig.: P. r. n. *Not* to be used over abraded portions of the skin.

INTERTRIGO
(Chafing).

This affection occurs with predilection in localities where opposed body surfaces rub against each other, and in the "napkin region." It is the result of irritation of the skin by acrid secretions or excretions (sweat, diarrheal stools, acid urine, purulent discharges, etc.), excessive heat or moisture. Intertrigo usually begins with simple erythema. At this stage it readily yields, in addition to removal of the etiologic factors, to the application of a dusting powder of:—

℞ Zinci stearatis	ʒiv	15
Bismuthi subnitratis	gr. xv	1
Amyli	℥j	30

and the separation of the apposed surfaces by thin layers of absorbent cotton. As the disease advances, the skin becomes glossy, moist, sticky, and denuded of epidermis, and the seat of papules, abscesses and ulcerations. In this condition intertrigo is very refractory to treatment, often demanding a complete change in the régime of the baby—beginning with its diet and ending up with its nurse. The customary daily tub-bath should be replaced by a sponge bath, taking special care to keep the affected parts of the skin perfectly dry. The denuded skin should once daily be painted with a 1 or 2 per cent. solution of nitrate of silver, and the entire diseased surface covered with the following ointment:—

Glossy, moist redness.

Change of diet or nurse.

Nitrate of silver.

℞ Acidi carbolici,		
Balsami Peruviani	āā *m* v	0.3
Olei lini,		
Adipis lanæ,		
Ung. zinci oxidi	āā ʒiv	15

Sig.: To be applied several times a day after carefully cleansing (with oil) and drying the affected parts.

PSORIASIS.

The disease is very exceptionally met in children under five years of age, but is not uncommon in older ones. It begins with minute white spots usually upon the extensor surfaces of the elbows and knees and upon the scalp, and gradually assumes the shape of disks with tawny-red base and silvery-white scales, not

Silvery-white scales on red base.

Probably parasitic.

rarely giving the skin the appearance of having been splashed with mortar. The cause of psoriasis being obscure (apparently of parasitic origin, though it seem to run in families), the treatment is necessarily symptomatic, and very unsatisfactory as to ultimate cure. Internally we may try small doses of arsenic, to be continued for several months, or thyroid extract. Externally we resort to alkaline baths, and, after removal of the scales, to an ointment composed of chrysarobin or salicylic acid and ichthyol.

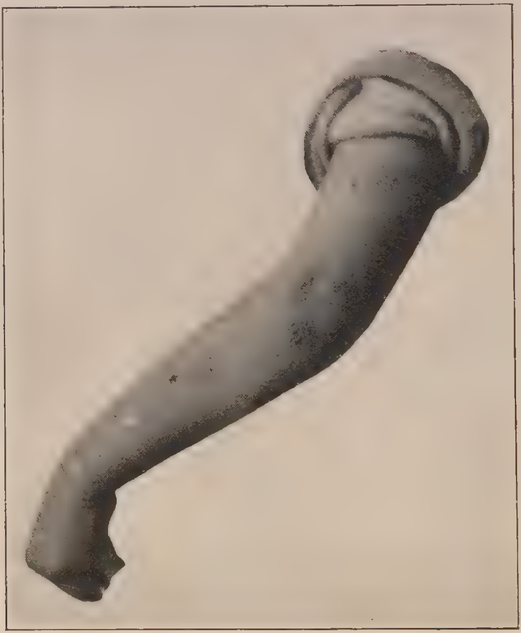

Fig. 200.—Psoriasis in a Girl Seven Years Old. (*Sheffield.*)

Arsenic.

℞ Acidi salicylici,		
Resorcini,		
Ichthyoli	āā ʒss	2
Ung. sulphuris	℥ij	60

Sig.: To be applied twice a day.

℞ Chrysarobini,		
Ichthyoli	āā ʒj	4
Ung. petrolati	℥ij	60

Sig.: To be applied once or twice a day.

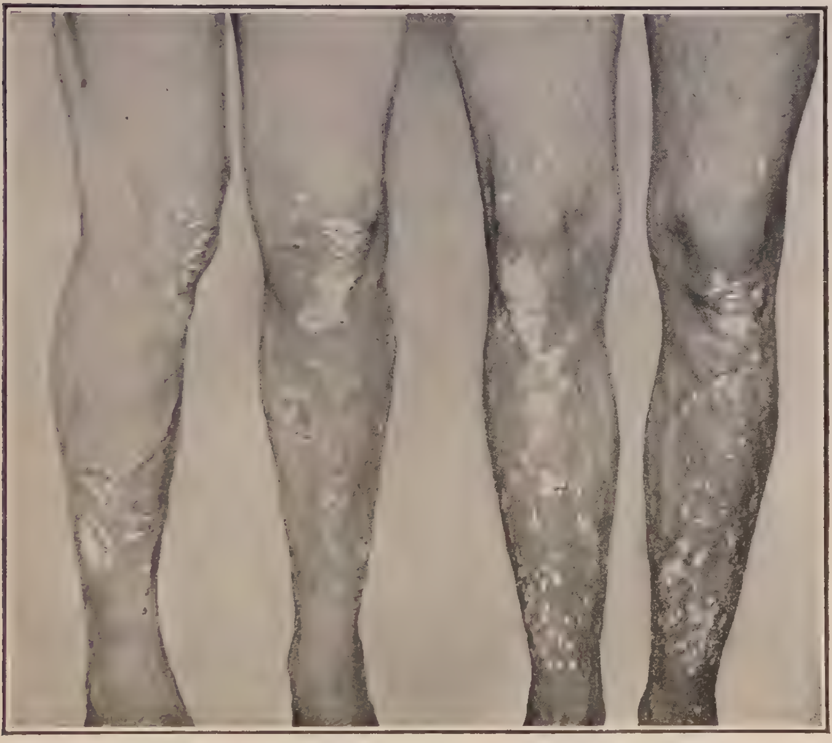

Fig. 201.—Psoriasis of the Legs. (*Shoemaker.*)

HERPES ZOSTER

(Shingles).

Contrary to what is observed in adults, herpes zoster in children is rarely accompanied by severe neuralgic pain. The eruption usually appears suddenly in the form of groups of vesicles along the tracts of either the intercostal or pudendal nerves, or the brachial plexus. The vesicles remain either isolated or coalesce and form large patches covered by yellowish-brown crusts. Different patches often exhibit different stages of development or decline. As a rule, the eruption is unilateral.

Vesicles along nerve tracts.

The course of the disease is usually completed within two weeks, except in cases leading to deep ulceration and sloughing (herpes gangrenosus), a very rare condition, usually the result of secondary infection. The treatment consists of local application

Danger of sloughing.

of a dusting powder or ointment composed of stearate of zinc with or without 2 per cent. of bismuth subnitrate or subgallate. Occasionally the nerve pain calls for some anodyne, *e.g.*, sodium salicylate.

Salicylates.

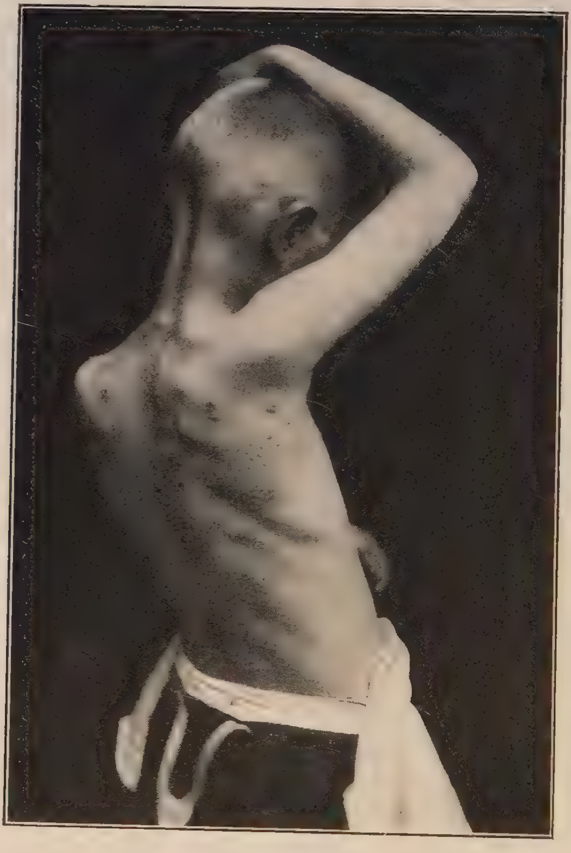

Fig. 202.—Herpes Zoster. (*Sheffield.*)

MILIARIA; LICHEN STROPHULUS
(Prickly Heat).

Harmless if not infected.

This very common affection in infants, especially during first dentition, appears suddenly upon the face, trunk and extremities either as discrete papules or vesicles from a pinhead to half a pea in size, or in groups upon a slightly reddened infiltrated base. It is produced by all sorts of external or internal irritations (heat, rough flannel underwear, overfeeding, etc.), and readily yields to attention to these causes, and the administration of mild laxatives. The slight itching may be relieved by alkaline or bran baths, and sponging of the body with Dobell's solution.

ECTHYMA

(Pseudofurunculosis).

Flat pustules surrounded by red zone.

It consists of pea- to bean-sized, flat pustules surrounded by a red zone. The lesions are situated chiefly upon the thighs, legs, shoulders and back and are frequently associated with eczema—probably produced by infection of the eczematous lesions during the act of scratching.

Ulcerations.

Gangrene.

Occasionally the pustules enlarge gradually and burst, leaving behind deep ulcers which heal very slowly with scar formation. These are prone to occur in ill-fed, scrofulous or otherwise seriously diseased children, and may sometimes end fatally as a result of gangrene of the skin.

Simple ecthyma usually responds to hot baths, antiseptic ointments, or sponging of the affected parts of the body with the following:—

℞ Etheris,
Tr. saponis viridis .... āā ℥j | 30

Large pustules should be treated by incision and antiseptic dressings. (See "Scrofulosis," page 370.)

IMPETIGO CONTAGIOSA.

The favorite seat of impetigo is the face, hands and scalp, but the other portions of the body are not exempt from inoculation.

"Stuck-on" appearance.

The eruption begins as small groups of minute vesicopapules which soon burst and dry up into yellowish crusts. When a crust has lasted for some time its surface becomes slightly lamellated and its edge detached, the crust then presenting the appearance as if "stuck on" to the healthy skin. The surface beneath the crust is raw and suppurating.

If further autoinoculation of the disease is prevented, impetigo usually heals spontaneously in about ten days. Otherwise, by the development of new lesions, it may persist for several weeks.

Contagious.

In view of the highly contagious nature of the disease and its tendency to run in epidemic form through schools or asylums, it is imperative to isolate all those children who are suffering from this disease and to employ active therapeutic measures to eradicate it.

This is readily accomplished by means of local antisepsis. After softening the crusts with warm carbolized sweet oil (1 per cent.), and removing them, and thoroughly washing the diseased surface with soft green soap, the spots are touched up with a 2 to 5 per cent. solution of nitrate of silver, and covered over with sterile gauze and adhesive plaster. This treatment is repeated for a few days and followed up with a 2 per cent. ichthyol in a 10 per cent. sulphur ointment.

Differentiation from simple impetigo.

Simple impetigo differs from the contagious variety by its lesions being pustular from the beginning and by showing no tendency rapidly to coalesce in large patches and to spread to other portions of the body. There is no history of contagion.

PEDICULOSIS CAPITIS

(Head-lice).

The favorite seat of the head-louse is the occipital portion of the scalp. In cases where the hair is thick and the parasites are few in number and hence not easily seen, their presence can readily be surmised by the existence of ova (nits), which are firmly attached to the shafts of the hair. The lesions produced by pediculi resemble those of eczema of the head—intense itching, pustules, scabs, matting of the hair, and marked enlargement of the glands of the neck.

Resembles eczema.

Isolation.

Children affected by pediculosis should be isolated for a few days until the disease is cured. The hair should be clipped, the scalp thoroughly cleansed with the tincture of green soap, and then dressed with a cloth dipped in petroleum or the tincture of larkspur (delphinium). A few such dressings usually suffice to effect a cure. After removal of the pediculi the scalp should be cleared of its remaining eruption by an antiseptic ointment.

PEDICULOSIS CORPORIS

(Body-lice).

Itching.

Body-lice are seldom seen in young children. They give rise to red dots, itching, and scratch marks. The diagnosis is settled by finding the parasite in the clothing or on the body of the child.

The treatment consists in destroying or baking the infested garments, scrubbing the child's body with green soap, and the application of a zinc and sulphur ointment until the eruption has entirely disappeared.

PEDICULOSIS PUBIS

(Crab-lice).

This skin affection is of diagnostic interest principally because of the power of the crab-louse to infest (in addition to the hair of the pubis, abdomen, chest and axilla) also the eyebrows and eyelashes, in the latter case giving rise to a clinical picture resembling blepharitis.

Infection of eyebrow and eyelashes.

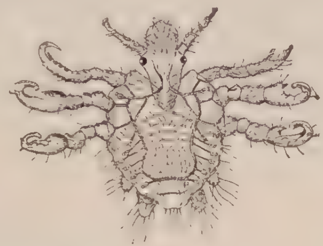

Fig. 203.—Phthirius Pubis [Crab-louse]. (After *Landois.*)

The insect succumbs rapidly to the effects of mercury ointment.

℞ Ung. hydrarg. nitratis .... ʒj | 4
Ung. petrolati .... ʒiij | 12
M. Sig.: Externally once a day.

SCABIES

(The "Itch").

The eruption of scabies is localized chiefly in places where the skin is thinnest, *i.e.*, the hands, the folds between the fingers, the flexor surfaces of the wrists, the anterior fold of the axilla, also the back and lower extremities. The characteristic skin lesion of scabies is an irregularly shaped, brownish-black ridge (cuniculus or furrow), the result of the burrowing process of the Acarus or Sarcoptes scabiei. The latter is the cause of scabies and can readily be demonstrated microscopically in the scrapings of the cuniculus. As the disease advances, it frequently spreads over the entire body and gives rise to a multiform eruption, consisting of papules, vesicles, pustules and hemorrhagic spots (scratch marks). It is accompanied by violent itching, which is worse at night, when the patient is warm in bed.

Localized chiefly where skin is thinnest.

Acarus scabiei.

Itching worse at night.

As the disease is highly contagious (conveyed through close

Isolation. **Disinfection of clothes.**

bodily contact, clothes, underwear and bedding), it is advisable to restrict the patient from too closely mingling with other members of the family or outsiders. The patient's clothes, bed-sheets, towels, etc., should be boiled, and the other unwashable articles thoroughly disinfected. Furthermore, all inmates of the house should be examined and, if necessary, treated for scabies, lest the disease will recur through renewed infection.

Green soap baths. **Sulphur.**

The treatment of scabies varies with the stage of the disease. Incipient scabies responds promptly to a few hot baths, thorough scrubbing of the affected skin with soft green soap and the inunction of sulphur ointment with 1 per cent. carbolic acid. The management of advanced scabies with the same therapeutic measures is not quite as satisfactory. A number of remedies (strong ointments of carbolic acid, naphthol, creolin, etc.) have been suggested for such cases, but owing to their irritating qualities (upon the skin and kidneys) should be used with caution. The following combination will probably be found to do well in the majority of cases:—

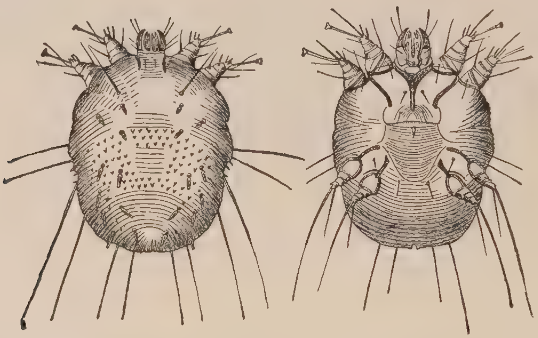
Fig. 204.—Sarcoptes Scabiei. Female seen from Above and Below. (After *Gudden.*)

℞ Mentholis,		
Pulv. camphoræ	āā gr. x	0.65
Olei cadini,		
Balsami Peruviani	āā ʒj	4
Ung. sulphuris	q. s. ad ℥ij	60

M. Sig.: To be applied in the evening after a hot soap bath.

℞ Acetanilidi	ʒss	2
Ung. zinci oxidi	℥j	30

M. Sig.: To relieve irritation.

TINEA TRICHOPHYTINA CAPITIS
(Ringworm of the Scalp, Herpes Tonsurans).

Highly contagious.

Ringworm of the scalp is due to the trichophyton fungus. It is highly contagious and often spreads with great rapidity and pertinacity in schools and children's homes where great numbers of inmates are crowded in comparatively small rooms.

Brittleness of hair.

The eruption consists of ring-shaped, slightly elevated, scaly, reddish, grayish, or greenish-yellow patches. The hair over the affected areas becomes brittle and loose and falls out, leaving behind bald and shiny spots.

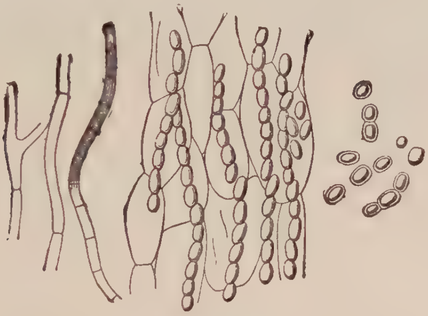

Fig. 205.—Trichophyton Tonsurans—Threads and Chains of Spores. × 400. (After *Bizzozero.*)

Tinea kerion.

At times the eruption is accompanied by severe local inflammation and exudation of a yellowish, viscid or gelatinous secretion—a condition generally described as *tinea kerion.*

Prevention of epidemics.

In the treatment of ringworm of the scalp it is essential not only to prevent spreading of the disease from one child to the other, but also autoinoculation from one part of the scalp to the other. This is best accomplished by sterilization (before and after using) of the hair clippers, scissors, combs, etc., and thorough scrubbing of the scalp with the tincture of green soap twice daily, and immediately after a haircut.

In an epidemic at an orphan asylum comprising nearly 400 cases of ringworm of the scalp, I found the following method of treatment exceedingly serviceable:—

℞ Acidi carbolici,		
Olei petrolei	āā ℥ij	65
Tinct. iodini,		
Olei ricini	āā ℥iiiss	110
Olei rusci (German)	q. s. ad ℥xvj	500

Specific method of treatment.

After clipping the hair close to the scalp this mixture is applied over the entire scalp—more thickly over the affected spots—by means of a painter's brush, once a day for five successive days. On the sixth day it is wiped off with a rag dipped in plain olive-oil; now the hair is clipped again and the scalp washed thoroughly but gently with green soap and a *soft* nailbrush, care being taken that all the scales and loose hair covering the scalp are removed. As a rule, no epilation is necessary. On the seventh day the mixture is reapplied as thickly as before and the whole process repeated regularly for three or four successive weeks—the length of time depending upon the severity of the case. New hair will now be found to appear, and no trichophyton fungi will be discovered in the hair epilated for microscopic examination.

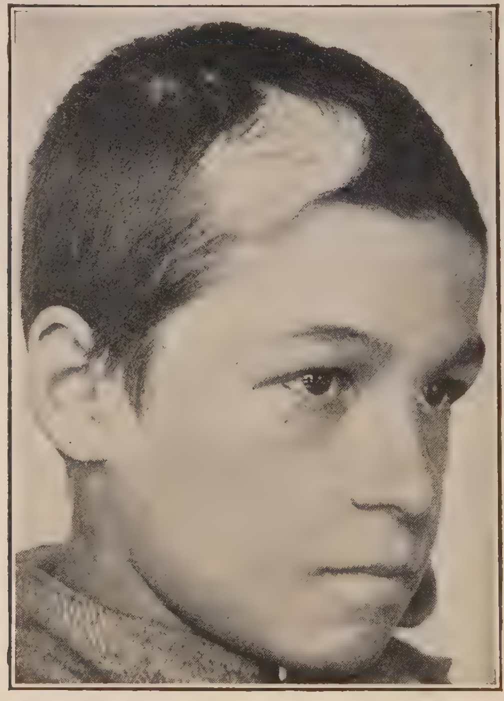

Fig. 206.—Tinea Tonsurans. (*Shoemaker.*)

These procedures are followed by a few days' application of a 10-per-cent. sulphur ointment, and then by the use of the following preparation for about two weeks:—

℞ Resorcini,		
Acidi salicyl.	āā ʒiv	16
Alcoholis	℥iv	120
Olei ricini	q. s. ad ℥xvj	500

This mixture considerably hastens the growth of the hair on the bald spots. In cases where isolation is impracticable or impossible, as often happens in private families, this resorcin mixture serves as an excellent substitute to prevent spreading of the affection.

Differentiation from tinea favosa.

Tinea tonsurans is not to be confounded with *tinea favosa,* a hair affection caused by the Achorion Schönleinii, and characterized by sulphur-yellow, cup-shaped crusts or scutula, penetrated by a hair or two.

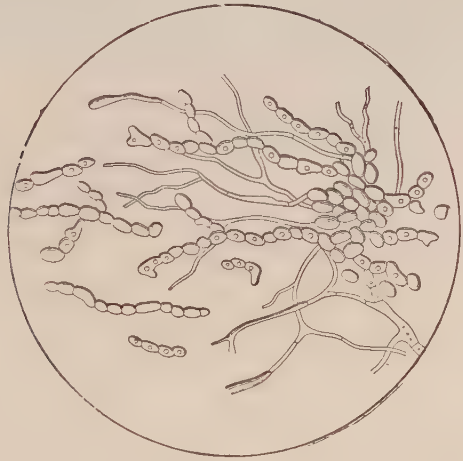

Fig. 207.—Achorion Schönleinii. × 400. (After *Bizzozero.*)

TINEA TRICHOPHYTINA CORPORIS

(Ringworm of the Body, Herpes Circinatus).

Ringworm of the body begins as a small, scaly, circular spot which rapidly spreads peripherally and clears in the center, resembling a "ring" in shape. The rings frequently coalesce, forming serpiginous lesions.

Tincture of iodin.

It is a trivial eruption and promptly yields to a few local applications of the tincture of iodin, white precipitate ointment, or glacial acetic acid (applied once every other day).

MOLLUSCUM CONTAGIOSUM.

Contagious molluscum is not rarely met in epidemic form in large institutions for children. The etiologic factor of the disease is as yet unknown.

Wart-like eruption principally on face.

The eruption appears principally upon the face, eyelids, neck and arms, and consists of discrete, semiglobular, waxy-white, umbilicated, small (up to a split pea) wart-like elevations, with sebaceous contents.

It is a benign affection and readily curable by ablation of the nodule or expression of its contents, and cauterization with tincture of iodin or 5 per cent. salicylic acid in collodion.

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bular Pneumonia.
mostly secondary.
ge. Under 3. Chiefly 2.
n delicate or debilitated children;
ause. In primary cases
ually pneumococcus.
secondary cases.
mixed infection –
Inf. process diffuse.
Onset – often gradual;
[illegible] [illegible], [illegible]
[illegible]
no typical course, generally
[illegible] by lysis.
Involves both lungs
sually. Lower, posterior
les.
Consolidation later.
gh none. Shallow TB
Usually.
Resolution slow. 1–2 mo.
[illegible] incomplete. Tendency
become chronic.
Relapses frequent.
Prognosis. Always
[illegible] [illegible] grave
accumulations –
– [illegible] – Empyema,
[illegible] [illegible]
[illegible].

Lobar Pneumonia
1. Most always primary.
2. Most common 3–4.
3. In healthy children.
4. Pneumococcus.
Micrococcus lanceolatus.
Diplococcus Frankel.
Weichselbaum –
5. Inf. process circumscrib
6. Onset sudden with
well-marked symp.
7. Typical course – Crisis
may be from 5–8 day.
8. One lobe or part. Left b
rt. apex. rt. base. left apex
9. Consolidation earlie
complete – sharply defined
10. Resolution rapid.
Usually complete within
week.
11. Rare.
12. No sequelae except
complications.
13. Prognosis. Good.
Rarely fatal except
from complications –
empyema, peri
meningitis.

ease -	Incubation	day of Rash	Character of R
easles	10-14 days	4th	Resembles flea b first appear o face + forehead, later [illegible] [illegible] extremities. [illegible] [illegible] [illegible] [illegible] [illegible]
et fever	[illegible] days, occasionally longer.	2nd day of fever.	Bright scarlet [illegible] [illegible] [illegible] [illegible] [illegible] [illegible]
cken-pox Varicella)	4-12 days	2nd day.	Small vesicl which do not become pustula
phoid fever.	10-14 days	7-14 days	Rose colored papu [illegible] [illegible] [illegible] [illegible] [illegible] disapp [illegible]
all-pox ariola)	~~10-14 days~~ 10-14 days.	~~7-14 days~~. 3rd day of fever.	Small [illegible] [illegible] [illegible] forming vesicle then pustules first appearing face and wrist

Rash dates	Incubation of disease	
7th day of fever	6-19 days 1- -10	generally 3 weeks
5th day of fever	5-7 days 3-7 [illegible]	
[illegible] of short duration	1-2 days.	
[illegible] in succession [illegible] 3 days [illegible]	21-30 days.	
1st day [illegible] 14th day [illegible]	14-21 days	

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